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THE LARYNGOSCOPE

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EAR - NOSE - THROAT

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THE
LARYNGOSCOPE.

VOL. XXXVI JULY, 1926 No. 7

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PRESENTATION OF MICROSCOPICAL SECTIONS OF VARIOUS PATHOLOGICAL CONDITIONS OF EAR.*

DR. FRANK A. BRIDGETT, Philadelphia.

Having attended the course in pathology of the ear, given by Prof. Otto Meyer, of Vienna, and having received the following slides for a permanent collection, I thought it would be of interest to the otologists to see the collection of the various pathological sections I possess.

Gentlemen: I will project two pictures of each slide on the screen side by side. One a lantern slide of the section, the other a picture projected from the Spencer microprojector of the actual section.

The object in projecting the lantern slide pictures is to show you the entire field, in order that you may become orientated in my description of the anatomy and, secondly, to point out the area I am describing.

The picture projected by the microprojector shows only a part of the field of the section the same as when viewed through the microscope.

SLIDE 1. ACUTE INFLAMMATION OF THE MIDDLE EAR.
Acute Otitis Media.

This is a picture of an acute otitis media. In studying the picture we note the following changes:

1. The greatly increased thickening of the drumhead as a whole.

*Lecture given by Dr. Frank A. Bridgett before the Philadelphia Laryngological Society, Feb. 2, 1925.
Slides obtained from Prof. Otto Meyer, of Vienna, given to students of his course.
Editor's Note: This mss. received in The Laryngoscope Office and accepted for publication March 4, 1926.

2. An increased thickening of the individual layers of the drumhead.

3. An enormous amount of purulent exudation in the tympanic cavity with some fibrin present.

4. An increased thickening of the mucous membrane on the inner wall of the tympanic cavity.

5. Little or no purulent exudation in the semicircular canals.

Taking up an analysis of the individual layers of the drumhead we find the changes as follows:

I. Skin Layer of the Drumhead.

1. The epidermis of the skin layer is thickened, most likely due to exudation.

2. The corium is thickened by edema, cellular infiltration, and an increased number of engorged blood vessels and capillaries are seen.

II. The Radial and Circular Fibrous Layers of the Drumhead.

The radial and circular fibrous layers of the drumhead are proportionately thickened, but no blood vessels or capillaries are seen in them, neither is there seen any cellular infiltration between their fibers.

The proportionate increase in their size is due to exudation of serum.

III. The Mucous Membrane Layer of the Drumhead.

1. Epithelium of the mucous membrane shows the individual cells swollen and enlarged.

2. There is not a distinct membrana propria seen, but the epithelium stains less deeply than the tunica propria beneath. No capillaries are seen in this layer.

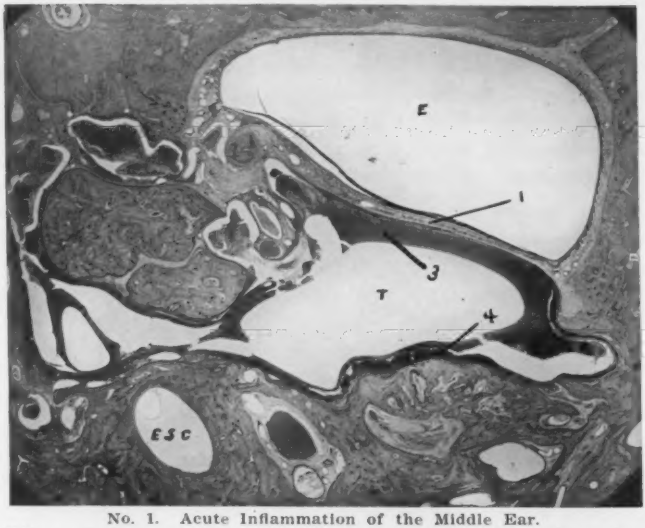
3. The subjacent tunica propria is greatly thickened by edema, cellular infiltration, and a great many engorged blood vessels and capillaries are seen.

Now what is the significance of this altered condition of the drumhead and the middle ear cavity with its contained secretion?

As Mallory in his work, "The Principles of Pathologic Histology," explains concisely what inflammation is I will quote freely from his work.

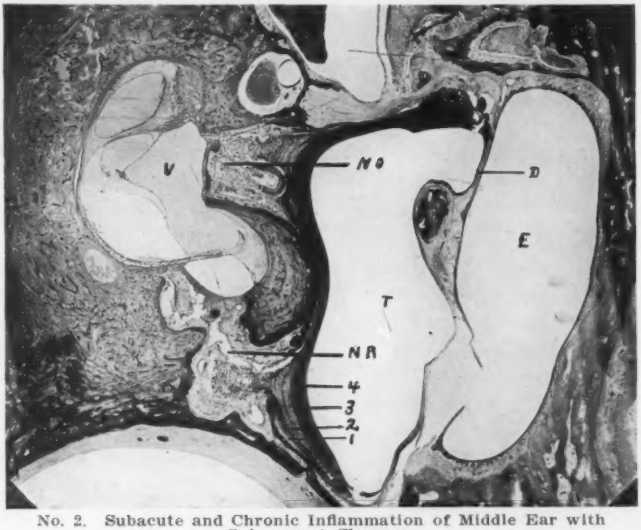
It means that we are dealing with an acute inflammation of the middle ear cavity and any inflammation is a reaction of a living organism to any injury done to it by injurious agents.

In the study of inflammation it is necessary to consider: 1. The injurious agent. 2. The reaction to the injurious agent. 3. The



No. 1. Acute Inflammation of the Middle Ear.

Slide 1. E. External auditory canal. T. Tympanic cavity. ESC. External semicircular canal. 1. Drumhead. 3. Purulent exudation. 4. Thickened mucous membrane.



No. 2. Subacute and Chronic Inflammation of Middle Ear with Subsequent Changes.

Slide 2. E. External auditory canal. D. Drumhead. T. Tympanic cavity. V. Vestibule. NO. Niche of oval window. N.R. Niche of round window. 1. Violet line of epithelial layer of mucous membrane. 2. Fibrinous layer. 3. Homogeneous necrotic layer. 4. Pus layer.

injury done to the emigrated cells and the fixed cells of the area invaded. *4.* The reaction to the injury produced.

1. The injurious agent. In the majority of the acute inflammations of the middle ear, the offending agents are usually bacteria.

2. The reaction to the injurious agent. The reactions to the injurious agents or bacteria are: *1.* Chemical; *2.* morphological: *3.* physiological.

1. The chemical reaction is manifested by an emigration of leucocytes from the blood vessels into the area in which the injurious agent is located and is due to toxins thrown off by the bacteria which exerts a chemotactic effect, and this chemotactic effect must be a chemical stimulus.

2. The morphological reaction is evidenced by the presence of: *1.* Plasma and fibrin; *2.* leucocytes; *3.* congested blood vessels.

3. The physiological reaction is seen in the impaired function of the part. The reason for the morphological reaction to the injurious agent or bacteria is: *1.* To get rid of it, if it is still present. *2.* To overcome its action so that it can no longer do damage.

As there are many more capillaries in nonuse than there are in use normally, and when we find an area invaded by an injurious agent, immediately the reserve capillaries are brought into play and we get an increased edema and an increased cellular infiltration.

The morphological elements called out of this excess circulation and the manner in which they respond are: *1.* Lymph (by exudation). *2. a.* Polymorphonuclear leucocytes (by emigration). *b.* Large mononuclear or endothelial leucocytes (by emigration). *c.* Lymphocytes (by emigration). *d.* Eosinophiles (by emigration). *e.* Mast cells (by emigration). *3.* Lymphocytes (by migration). *4.* Red blood cells (by diapedesis).

Taking up the morphological elements in sequence we have:

1. Exudation. The varieties of acute inflammatory exudation are: *1.* Serous exudation; *2.* fibrinous exudation; *3.* purulent exudation or suppuration; *4.* hemorrhagic exudation; *5.* catarrhal exudation.

The kind we have to deal with in this picture is purulent exudation, and what do we find?

1. Plasma and lymph are present, manifested by swollen appearance of drum.

2. Leucocytes, especially the polymorphonuclear, are in the preponderance.

3. Little fibrin is seen.

Purulent exudate is called pus, because it is dead leucocytes in this plasma and lymph that forms pus.

2. Emigration of Leucocytes. In an acute inflammation, the first cells to emigrate are the polymorphonuclear leucocytes and they travel towards the site of the injury. Emigration through vessel walls takes place not only from vessels acted upon by the injurious agents, but also from other vessels in the immediate neighborhood.

Taking up the part played by the various leucocytes seen in emigration we find:

No. 3. Chronic Otitis Media.
Slide 3. E. External auditory canal. T. Tympanic cavity. N. Newly organized tissue. S.S. Sacs. V. Vestibule. D. Tympanic membrane.

A. Polymorphonuclear Leucocytes.

1. The polymorphonuclear leucocytes play the most important part in acute inflammation.

2. They are the first to emigrate.

3. They are the only ones present in the early stages.

4. They invade masses of bacteria.

5. They incorporate some of them.

6. They do not multiply by mitosis in the tissues like other leucocytes.

7. They are called out in great numbers by pus cocci.

8. They never incorporate other cells.

B. Endothelial Leucocytes.

1. Little in evidence early in acute inflammation.

2. Appears later when the polys are diminishing in numbers, and the injurious agent is weakened in strength.

3. They are called out by mild injurious agents which act for a considerable length of time or are mildly toxic, or act mechanically only.

4. They form the chief or only cell reaction to leprosy bacillus, typhoid bacillus, and usually to the tubercle bacillus.

5. They attend: to most foreign bodies; to carbon; to free fat in chronic lesions; to cholesterin crystals.

6. They form: foreign body giant cells; giant cells in tuberculous lesion; giant cell sarcoma.

7. They are phagocytic for certain bacteria and for other leucocytes.

8. They multiply by mitosis.

C. Lymphocytes.

1. These cells reach the injured area both from the blood and lymph vessels by emigration and migration.

2. They multiply by mitosis in the tissue.

3. They are never phagocytic for bacteria or cells.

4. Some undergo change in the tissue and are called plasma cells.

Lymphocytes are most abundant after the inflammation has lasted several days and are most numerous in exudative processes which have persisted for several weeks.

When a lesion has healed, lymphocytes persist for a long time in the lymphatics leading away from the afflicted tissue. The chief function of the lymphocytes seems to be to neutralize or dispose of injurious substances which are being absorbed through the lymphatics.

D. Eosinophiles.

They are found in very small numbers, as a rule in acute inflammations, but usually are more numerous during the stage of repair.

They are usually found more numerous in chronic lesions.

1. Cancer of cervix uteri.

2. Hodgkin's disease.

3. Trichiniasis.

Their function is unknown, probably to counteract certain injurious substances.

3. Migration.

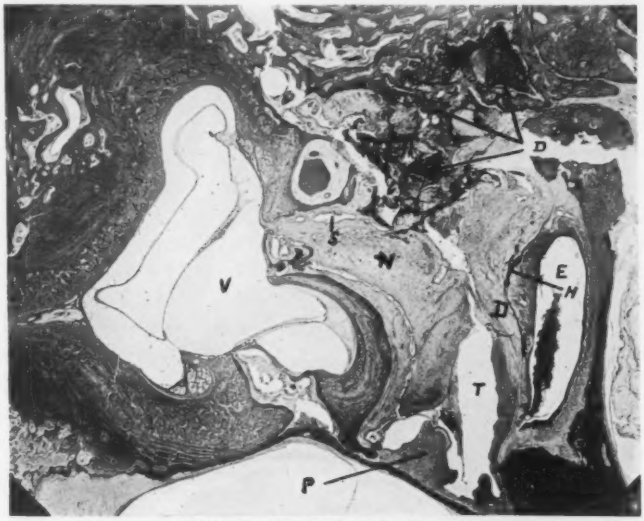
1. The lymphocytes emigrate and migrate to a lesion from the blood and lymph vessels.

2. They multiply by mitosis.

3. They are never phagocytic for micro-organisms or cells.

4. Diapedesis.

In the capillaries not only the leucocytes, but also the red blood cells pass through the vessel walls. The red blood cells escape passively through the breaks in the vessel walls, usually after the emigration of the leucocytes, and cause a small hemorrhage.



No. 4. Otitis Media, Meningitis (Influenza and Streptococcus Mucosus).

Slide 4. E. External auditory canal. D. Drumhead. H. Hemorrhagic exudate in fibrous layers of tympanic membrane. T. Tympanic cavity. N. Newly organized tissue in tympanic cavity. S. Sac. P. Polyp. V. Vestibule. D. Destruction of bone.

3. The Injury Done to the Emigrated Cells and the Fixed Cells of the Area Invaded.

In an acute inflammation the injurious agent, in this case bacteria, throws off a substance which produces a chemical stimulus and a toxic effect.

a. The effect produced on the mobile cells.

The response to the chemical stimulus on the mobile cells is an emigration of the white cells of the blood through the vessel walls,

especially the capillaries to the area invaded. As a result of their passage through the vessel walls, the endothelial plates are separated and there is a passive exit of the arythrocytes through the same avenues of exit. This passage is a passive action, and takes no part in the production of it. This is called diapedesis and, of course, we find a hemorrhagic condition as a result.

The white cells on arrival at the invaded area engage the bacteria in battle. Now the toxin thrown off by the bacteria begins to exert its effect, and as a result the white corpuscles are injured or killed, at the same time the bacteria are being destroyed and as a result deleterious substances from the dead leucocytes and the dead bacteria are thrown off and seriously effects to the extent of destruction of the fixed cells in the area in which the battle takes place.

It resembles a battlefield in which two armies are opposed, with dead and dying on both sides and the destruction of the area in which both are opposed. The presence of dead white corpuscles in exudate means pus.

b. The effect produced on the fixed connective tissue cells of the invaded area.

Pus always destroys the fixed connective tissue cells in which it comes in contact, and later when the cells are necrosed or dead, solution takes place.

The tissue solution or dissolving effect in suppuration is generally due to the action of ferments secreted by the polymorphonuclear leucocytes, acting upon the dead cells attracted by the chemotactic effect or chemical stimulus of the bacteria.

4. The Reaction to the Injury Produced or Repair.

If the action of the bacteria has been overcome the fixed connective tissue cells begin to proliferate to replace in kind the cells destroyed. This proliferation is stimulated by the presence of fibrin, which is generally present more or less in the exudative material found in the area destroyed.

SLIDE 2. SUBACUTE AND CHRONIC INFLAMMATION OF MIDDLE EAR WITH SUBSEQUENT CHANGES.

Older Stage of an Acute Otitis Media.

This picture is an older stage of an acute otitis media than the preceding picture.

A study of this picture shows the following changes:

1. There is very little swelling or infiltration of the drumhead as a whole.

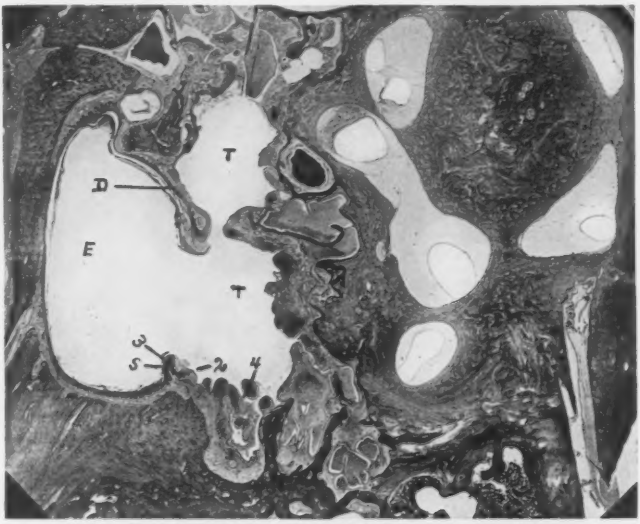
2. The modified skin layer, the epidermis and corium, has undergone very little change, little or no cellular infiltration in its tissue.

3. The radial and circular layers are slightly thickened, probably due to the plane of the cut.

4. The mucous membrane layer shows a slight swelling with a moderate amount of cellular infiltration. The greatest changes have taken place on the inner wall of the tympanic cavity, especially in the niches of the oval and round windows.

Here we see four layers, namely: *a.* Mucous membrane layer; *b.* fibrinous layer; *c.* a homogeneous necrotic layer; *d.* pus layer.

Taking up the study of the inner wall we find a very interesting condition, namely:



No. 5. Epidermization of Margins of Perforation (Chronic Suppurative Otitis Media).

Slide 5. E. External auditory canal. D. Drumhead. T. Tympanic cavity. S. Stump of drumhead. 2. Line of demarcation of epidermis and epithelium. 4. Polyp. 3. Evidence of calcification at tip.

1. A thin violet line differentiating a greatly thickened mucous membrane from a newly semiorganized tissue resting upon it and resting upon this semiorganized tissue we find a homogeneous layer of necrotic tissue, and on top of this layer is seen a layer of cellular pussy exudate.

Making a study of the mucous membrane first, we find the tunica propria moderately infiltrated with leucocytes, the blood vessel capillaries are quite numerous and filled with blood.

The violet line seems to be a thinned-out, flattened, single layer of epithelial cells.

2. Resting on top of this is a semiorganized fibrinous exudate moderately infiltrated with leucocytes, and in the niches of the round window and oval windows banked up against the secondary membrane and the footplate of the stapes we find this fibrinous exudate more highly organized by the ingrowth of new, young fibroblasts or young connective tissue cells. Between the mucous membrane and this newly organized tissue we find areas that correspond to sacs. This tissue is sparingly infiltrated with leucocytes.

No. 6. Granulation Polyp of the Ear.
Slide 6. S. Surface devoid of epithelium.

We know we have newly organized tissue formed for the reason that

1. We have the presence of sacs.

2. It is lying on top of the mucous membrane and we see an ingrowth of young fibroblasts.

3. There are capillaries filled with blood in this tissue.

4. The tissue is more loosely arranged than the underlying denser mucous membrane.

5. In this loose tissue we find a great deal of plasma and lymph.

This is the fibrinous type of acute inflammatory exudation because:

1. It consists of tissue which has an abundance of fibrin, a thin network of tissue with lines showing the nodal points. *2.* This type

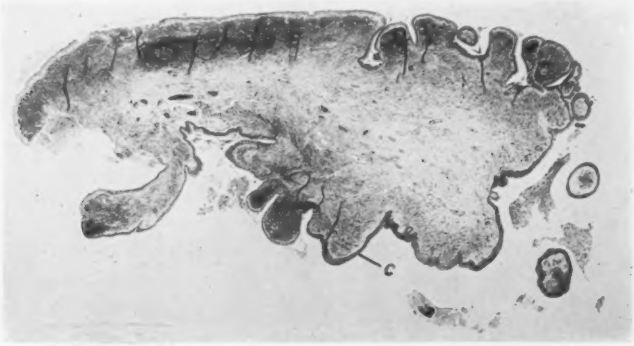
of exudation occurs most frequently on serious and epithelial-lined surfaces. *3.* Fibrinous exudation is a direct stimulus to the growth of the fibroblasts, with its fibroglia and collagen fibrils.

3. We find a homogeneous layer on top of the fibrinous area which is necrotic, showing that this is the simple fibrinous type of exudation as we see that the fibrin is greatly in excess of the necrosis.

4. The fourth layer resting on the necrotic layer is composed of various types of leucocytes, the polymorphonuclears predominating.

SLIDE 3. CHRONIC OTITIS MEDIA.

This picture is a later stage of an otitis in which young fibroblasts, and their fibroglia are seen in newly organized tissue. The capillaries are quite numerous in this tissue.



No. 7. Polyp of Ear Showing Columnar Epithelium.
Slide 7. C. Columnar epithelium.

The open areas are sacs between the mucous membrane of the tympanic cavity and the newly organized tissue.

There must have been a great deal of fibrin in this lesion for the reason that there is such an extensively newly organized tissue present. In this tissue is seen young fibroblasts and vascular endothelium in the form of capillaries.

At the places where the sacs touch the mucous membrane lining the walls of the tympanic cavity, is seen the places of ingrowth of the fibroblasts from the tunico propria of the mucous membrane, and there also is seen the epithelial cells growing across these bands of attachment to line the tissue on the newly organized side. Floating in the sacs is seen masses of leucocytes.

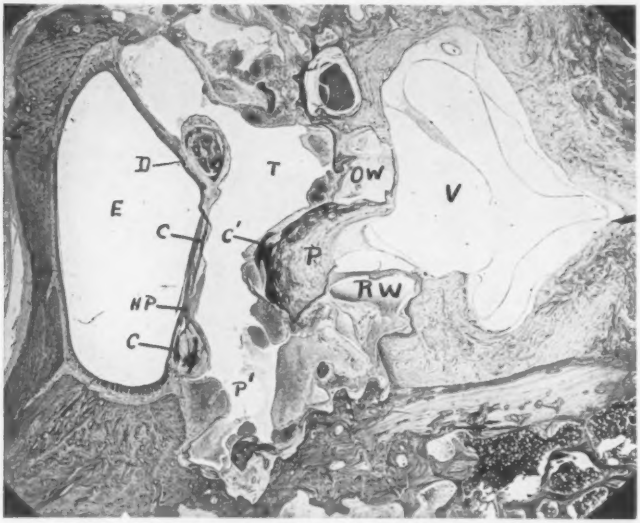
This organized mass extends from the footplate of the stapes across the tympanic space to become attached to the malleus handle,

and if we note carefully we see that the drum is greatly retracted. This case in life would show a severe physiological reaction to the injurious agent, namely an impairment of the conductive apparatus.

The labyrinth is practically free from exudation and cellular infiltration.

Slide 4. Otitis Media, Meningitis (Influenza and Streptococcus Mucosus).

This is a vertical section, the plane of which is at right angles to the long side of the petrous portion of the temporal bone, passing



No. 8. Calcification of Drumhead and Inner Wall of the Tympanic Cavity.

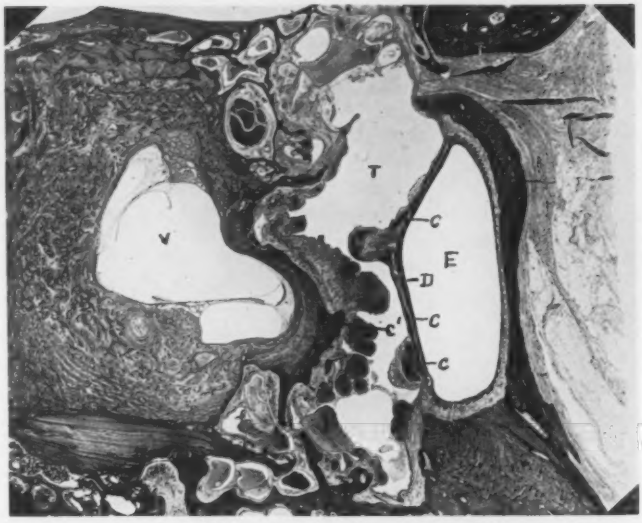
Slide 8. E. External auditory canal. D. Drumhead. T. Tympanic cavity. P. Promontory. R.W. Round window. O.W. Oval window. V. Vestibule. C. Calcification in tympanic membrane. C. Calcification of promontory. H.P. Healed perforation. P. Polyp.

through the vestibule, the tympanic cavity and the anterior wall of the external bony canal.

The first thing we note is the thickness of the drumhead, making due allowance for the plane of the cut.

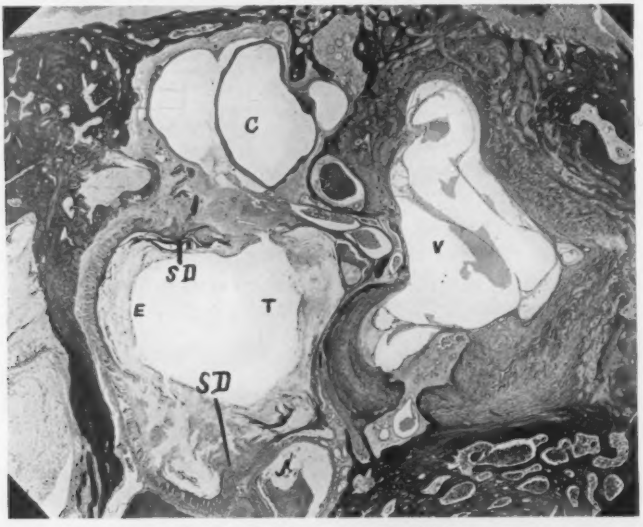
2. There is seen newly organized tissue practically obliterating the entire tympanic cavity.

3. The mucous membrane of the tympanic cavity is greatly thickened, in some places to the extent of polyp formation.



No. 9. Calcification of Drum and Promontory.

Slide 9. E. External auditory canal. D. Drumhead. T. Tympanic cavity. V. Vestibule. C.C.C. Calcification in tympanic membrane. C. Calcification of promontory.



No. 10. Cholesteatoma in Attic.

Slide 10. E. External auditory canal. T. Tympanic cavity. V. Vestibule. S.D. Stumps of tympanic membrane. C. Cholesteatoma in attic.

4. At the top there has been a great destruction of bone, but the periosteum lining the septa is intact.

Taking up the various areas separately we find:

1. The drumhead. There seems to have been a great stimulation of the cells of the fibrous layer, as they are seen growing out into the corium of the skin and growing into the mucous membrane. At the same time there is seen a retrograde process of a hyaline degeneration all through this area, practically the whole process is a hyaline degeneration.

In the fibrous layer of the drum is seen a hemorrhagic exudate extending downward. At one spot is seen a hemorrhagic bleb that has separated the corium from the radial fibrous layer.

2. The tympanic cavity. The newly organized tissue in the tympanic cavity is separated from the walls by many sacs. Of course it has all the elements of newly organized tissue.

3. The mucous membrane is greatly thickened to the extent of polyp formation.

4. The area at the top shows great destruction of bone, with an intense diapedesis together with hyaline degeneration. The labyrinth is apparently unaffected by this hemorrhagic exudate, although the patient died of meningitis. This condition is due to the influenza bacillus of Kitasato primarily, and secondarily to the streptococcus mucus.

According to Politzer: *1.* Bacteriologically. The primary infection is due to the bacillus influenza (la grippe) of Kitasato.

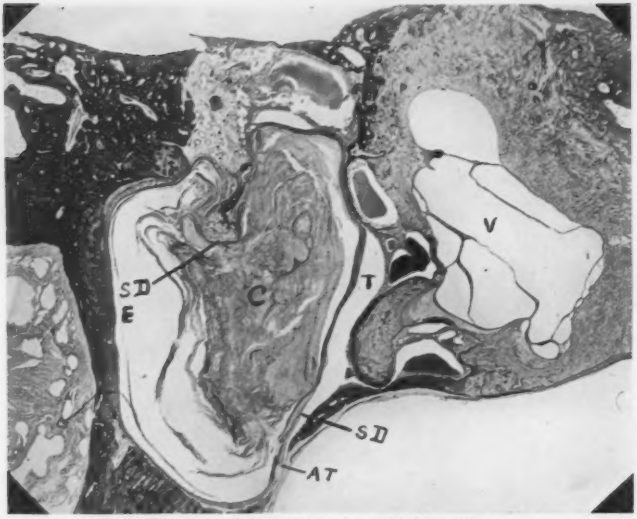
The primary bacteria giving rise to the affection are soon destroyed by the micro-organisms of the secondary infection, which in this case was the streptococcus mucosus.

Cases of otitis due to streptococcus mucosus has a tendency to heal rapidly in the tympanic cavity, but during a subsequent latent stage to extend to neighboring parts (mastoid process, cranial fossa).

Simultaneous involvement of the labyrinth is very rare in influenza otitis.

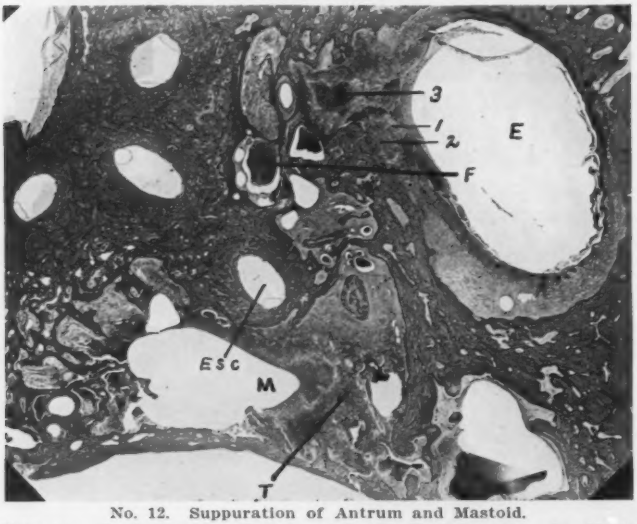
Alexander states: The influenza bacillus causes hemorrhagic abscesses. The streptococcus mucosus produces a mucus pus of slight fluidity. What is streptococcus mucosus?

Richardson has reported an organism found in lobar pneumonia, cerebrospinal meningitis and mastoid disease bearing resemblance to both pneumococci and streptococci—streptococcus capsulatus, other names are streptococcus mucosus and penumococcus mucosus.



No. 11. Cholesteatoma of Middle Ear.

Slide 11. E. External auditory canal. S.D. Stumps of tympanic membrane. C. Cholesteatoma. A.T. Annulus tympanicus. T. Tympanic cavity. V. Vestibule.



No. 12. Suppuration of Antrum and Mastoid.

Slide 12. E. External auditory canal. F. Facial nerve. M. Mastoid. E.S.C. External semicircular canal. 1. Epidermis growing into tympanic cavity. 2. Partially organized tissue. 3. Purulent exudate. T. Partially organized tissue.

SLIDE 5. EPIDERMIZATION OF MARGINS OF PERFORATION (CHRONIC SUPPURATIVE OTITIS MEDIA).

This picture shows the results of an old chronic discharging ear, as evidenced by the following anatomical and pathological changes:

1. The drumhead is perforated with a loss of tissue.

2. With the loss of tissue the epidermis of the modified skin layer of the drum has grown over the edge and into the tympanic cavity, apparently displacing the epithelial layer of the mucous membrane. This proliferation has extended downward on the drum to the floor of the tympanic cavity, where it comes to a stop at the junction with the epithelial layer of the mucous membrane in which we see a sharp line of demarcation.

The epidermis is five times as thick as the epithelial layer of the mucous membrane at this junction.

3. In the tip of the short side of what remains of the drum is seen a bony area, showing evidence of calcification and ossification as a result of a purulent process.

4. The mucous membrane is enormously thickened and polypous, with creases between the polyps, acting as wonderful retention recesses in a moist, warm, unaerated, undrained tissue and extremely favorable to the growth of bacteria. One large area in the center is more densely organized than the others, with a moderate amount of leucocytic infiltration.

On the inner wall and as a matter of fact on the entire tympanic cavity we see the mucous membrane enormously thickened, with a great amount of leucocytic infiltration.

The epithelial layers at all points are intact, and not exfoliated. Very little congestion of the blood vessels or capillaries is seen.

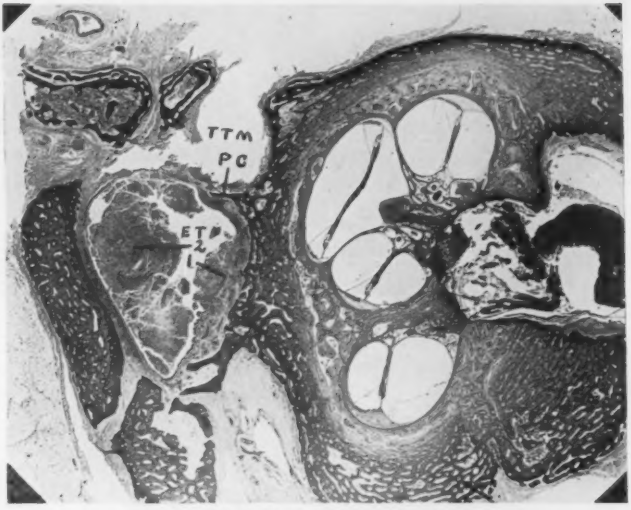
Of course the thickening of the mucous membrane is due to serous exudation, with no fibrin seen at any point. Of course, we readily see why it is almost an impossibility to cure a condition of affairs like this for the following reason:

1. The perforation of the drumhead cannot be filled in because the edges are epithelialized.

If the perforation is small and we cauterize it we destroy the epidermis and granulation tissue forms which is connective tissue of the corium and it fills the gap before the epidermis can prevent its growth, having filled in the gap, then the epidermis covers it and the drum is intact and the perforation absent.

SLIDE 6. GRANULATION POLYP OF THE EAR.

When an epithelial surface has been destroyed extensively, the only tissue present is connective tissue and vascular endothelium,



No. 13. Diphtheria of Eustachian Tube.

Slide 13. T.T.M. Compartment of tensor tympani muscle with greater part of muscle lost in preparation. E.T. Eustachian tube. 1. Mucous membrane destroyed. 2. Purulent exudation.

No. 14. Scarlatina of Eustachian Tube.

Slide 14. E.T. Eustachian tube. 1. Necrosis. 2. Fibrin.

and as we find fibrin on the surface and as fibrin is a direct stimulus to connective tissue, we get an increase growth of this tissue which proliferates faster than the epithelial covering, we, therefore, have a granulation polyp.

In this tissue we find a vast amount of leucocytes, together with a great amount of vascularity and the covering devoid of an epithelial layer. This tissue grows out through a perforation from the mucous membrane.

SLIDE 7. POLYP OF EAR SHOWING COLUMNAR EPITHELIUM.

This is a true polyp of the ear, as it springs from mucous membrane that is lined by columnar epithelium. Two principal forms are generally found, the round celled polyp and the fibromata.

This is the round cell form in which we see that the tissue of the body is loose, and filled with an abundance of round cells, together with a great many congested blood vessels. The round cell infiltration is extremely great on the surface of the polyp. The surface presents gland-like depressions into the body of the polyp and the surface is covered by columnar epithelium.

SLIDE 8. CALCIFICATION OF DRUMHEAD AND INNER WALL OF THE TYMPANIC CAVITY.

Here is seen a calcified area on the drumhead and on the inner wall of the middle ear. Taking up a study of the drumhead we see an area that must have been a perforation on the inferior portion, but which has been healed by the proliferation of the cells of the epidermis, which has completely filled in the gap, and the proliferation of the cells of the epidermis has extended superiorly and inferiorly on the inside of the drumhead for a considerable distance.

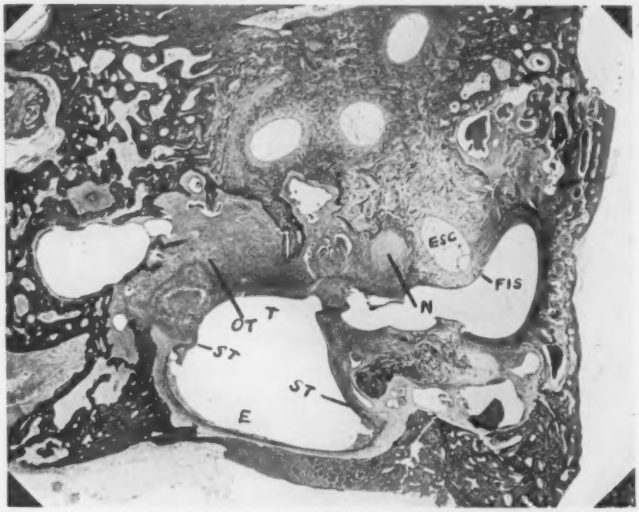
In the healing process we don't see the replacement of the component parts, namely, the radial and circular fibrous layer or the mucous membrane.

Throughout the tympanic cavity we see a greatly thickened mucous membrane polypus in type at places, also there is seen newly organized tissue in this cavity, together with a great deal of leucocytic infiltration.

Between this newly organized tissue and the mucous membrane lining we see areas which are sacs.

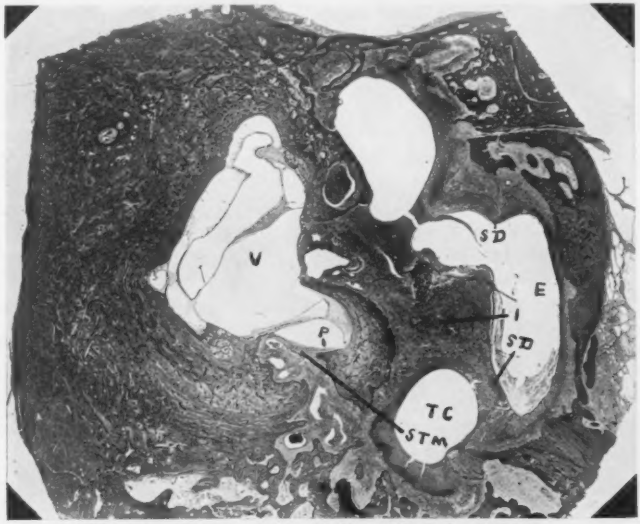
In the radial and circular fibrous layers of the drumhead there is seen a deep purple stain, which indicates that we have a deposition of lime salts, or pathological calcification.

Looking at the inner wall of the tympanic cavity we see an area over the entire promontory in which the mucous membrane is greatly



No. 15. Chronic Otitis Media, with Fistula or External Semicircular Canal. Diffuse Purulent Labyrinthitis.

Slide 15. E. External auditory canal. S.T. Stumps of membrane tympani. O.T. Newly organized tissue in tympanic cavity. T. Tympanic cavity. N. Facial nerve. E.S.C. External semicircular canal. F.I.S. Fistula in external semicircular canal.



No. 16. Chronic Otitis Media with Labyrinthitis.

Slide 16. E. External auditory canal. S.T. Stumps of membrana tympani. T.C. Tympanic cavity. S.T.M. Secondary tympanic membrane. V. Vestibule. P. Purulent exudate resting on the secondary tympanic membrane. 1. Newly organized tissue in tympanic cavity.

thickened, together with a greatly increased thickening of the periosteum, which, of course, is dense fibrous and fibro-elastic connective tissue normally, but a change has taken place, most likely due to a purulent process, and we see an area in which the fibrous cells have greatly proliferated, and they have become compressed, and it has the same appearance as the fibrous layers of the drum, and it is this changed fibrous area that has become impregnated with lime salts.

This fibrous area has undergone hyalinization. It is possible that this case was tubercular for the reason as later described.

1. Now, what is the significance of this calcification of the drumhead and promontory?

Gideon Wells (pathological calcification and ossification) states that the essential differences of pathological calcification and normal ossification are:

PATHOLOGICAL CALCIFICATION.	NORMAL CALCIFICATION.
1. Deposits of lime salts take place in dead tissue not infected and not absorbed, that has suffered a partial or total loss of vitality and that has become hyaline.	*1.* Deposits of lime salts take place in a specialized tissue of low vitality.
2. Lime salts are deposited in clumps.	*2.* Lime salts are deposited in an even manner throughout the tissue.
3. Deposition of lime salts take place in *any* kind of degenerated tissue.	*3.* Deposition of lime salts take place only in certain varieties of connective tissue.
4. Calcium phosphate is in the preponderance.	*4.* Calcium phosphate is in the preponderance.
5. Deposition of lime salts never occur in normal tissue.	*5.* Deposition of lime salts occurs in normal tissue in the formation of bone.

Order of calcification:

1. Necrotic masses (eye).

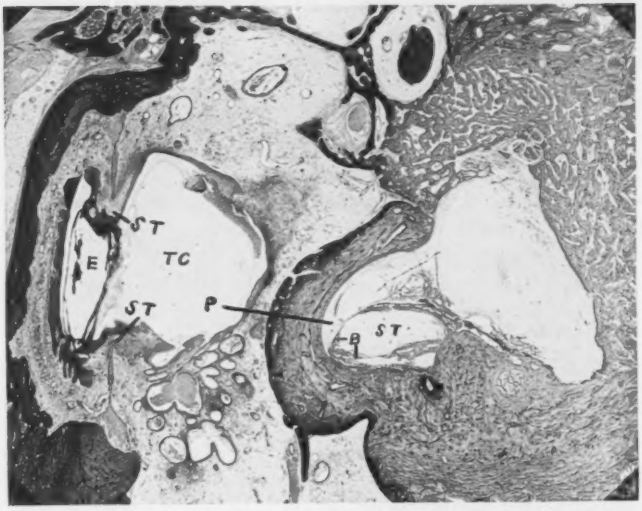
2. Scar tissue that has become hyaline subsequent to shutting off the circulation by contraction of tissues about the blood vessels.

3. Elastic tissue as internal elastica, that is why we get calcareous deposit and atheromata in arteriosclerosis.

4. Calcification of epithelium does not occur, but when it does is always preceded by a hyaline change, in which the calcium is deposited later (epithelial pearls).

Etiological factors in the causation of pathological calcification:

1. Calcium salts must come from the blood where they are held in solution by the proteids as carbonates and phosphates. This is



No. 17. Labyrinth Suppuration and Later Organization of the Same.

Slide 17. E. External auditory canal. S.T. Stumps of membrana tympani. T.C. Tympanic cavity. S.T. Scala tympani. P. Pectinate ligament. B. New bone near floor of scala tympani and also in pectinate ligament.

No. 18. Atrophic Labyrinth.

Slide 18. C. Corti's organ. 1. Break in lamina spiralis ossea at junction of modeoleus.

an unstable condition, because of the small proportion of calcium in the blood (about 1/10,000) and, therefore, capable of being overthrown either by an: *1*. Increased alkalinity. *2*. Changes in the proteids. *3*. Changes in the quantity or composition of the calcium salts.

2. Retrogressive changes are required: *1*. Hyaline degeneration. *2*. Often following inflammatory changes the collagen fibrils of connective tissue are rendered invisible, as they usually are in cartilage and bone, probably in the same way by a deposition of a homogeneous substance between them which binds them together; this is found frequently in the ear and healed tuberculous lesions.

3. The circulation is very feeble in areas that are to become calcified, no red cells being present to permit of oxidative changes.

4. Tissue juices seeping through slowly and the salts being precipitated. The most plausible idea for the deposition of calcium salts in area of tissue degeneration is the one in which there are formed in the degenerated area calcium binding substances, especially phosphoric acid, since it is phosphate that most of the calcium is bound to and the possible sources of phosphoric acid is seen in nucleoproteids and lecithin.

Relation of fatty acids to calcification: Fatty degeneration is calcified and calcification is rare without fatty degeneration.

Examples: Caseous tubercles are almost invariably calcified when healed; while gummas are relatively little affected.

Slide 9. Calcification of Drum and Promontory.

This picture shows an interesting condition. We see in the fibrous layers of the drum areas of calcification, also on the promontory are seen areas of calcification confined exclusively to the periosteum, and at one spot is seen evidence of necrosis.

The description of the preceding slide is applicable here.

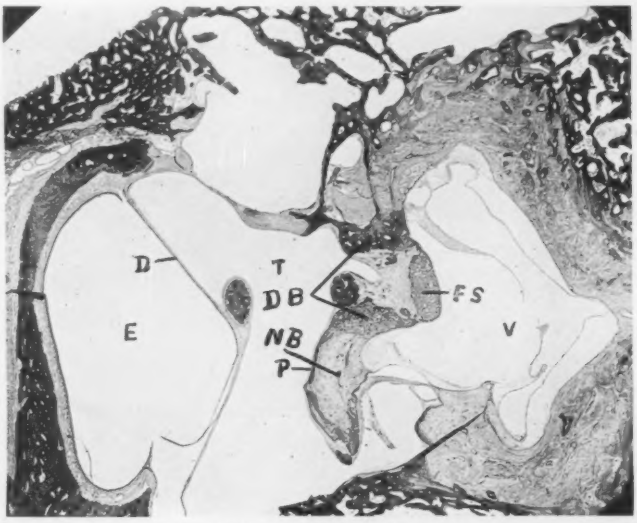
Slide 10. Cholesteatoma in Attic.

A cholesteatoma is an excessive production and desquamation of epithelial cells growing into the tympanic cavity through a perforation from the skin lining the external auditory canal.

The necessary factors to produce this condition are: *1*. A chronic discharging ear. *2*. A perforation in the drumhead in Shrapnell's membrane. *3*. The mucous membrane of the tympanic cavity being partially or wholly denuded of its epithelial covering.

The causes advanced are many, but the most likely cause is with perforations of pars flaccida; the invasion takes place the more easily, because a more highly developed band of cutis and epidermis

No. 19. Mastoiditis, Labyrinthitis, Suppurative Meningitis.
Slide 19. P. Purulent exudate in scala tympani.



No. 20. Otosclerosis.
Slide 20. E. External auditory canal. D. Drum membrane. T. Tympanic cavity. D.B. Diseased bone around oval window. P. Promontory. N.B. Normal bone. F.S. Footplate of stapes diseased. V. Vestibule.

extends from the superior wall of the external canal down over the wall of the tympanic membrane.

Now look at the slide and we find the drum gone with only a stump left on each side and over the edges we find the same type of skin tissues (thickened epiderm and corium) growing into the tympanic space on a saccule. There are seen in the tympanic space about two saccules and over these the epithelium is found.

It seems as if the connective tissue of the corium of the skin mingles with and grows on top of the tunica propria of the mucous membrane in which the epithelial surface has been lost and that is why the skin epithelium is found growing in large quantities in a mucus lined cavity. It produces a necrosis and absorption and atrophy as result of pressure and may invade vital structures. The interior of mass is purulent, foul and cheesey, and contains cholesterin crystals (▟) syringe ear and examine for crystals.

The symptoms according to Politzer are headaches, pain over temporal region, and dizziness as result of pressure on stapes and other associated symptoms:

Terminations: *1.* Perforation into the vestibule or any of the canals or into the internal meatus.

2. Death: *1.* Through pyemia of the septic degeneration of secretion behind the cholesteatomatous mass. *2.* Meningitis. *3.* Brain abscess. *4.* Erosion of sigmoid sinus or jugular fossa, with sinus phlebitis.

According to Gleason no operative procedure can be expected to produce a permanent cure of chronic otorrhea due to cholesteatoma, due, perhaps, to the fact that cholesteatoma prolongations force their way into the Haversian canals, thus forming centers from which it grows again.

Slide 11. Cholesteatoma of Middle Ear.

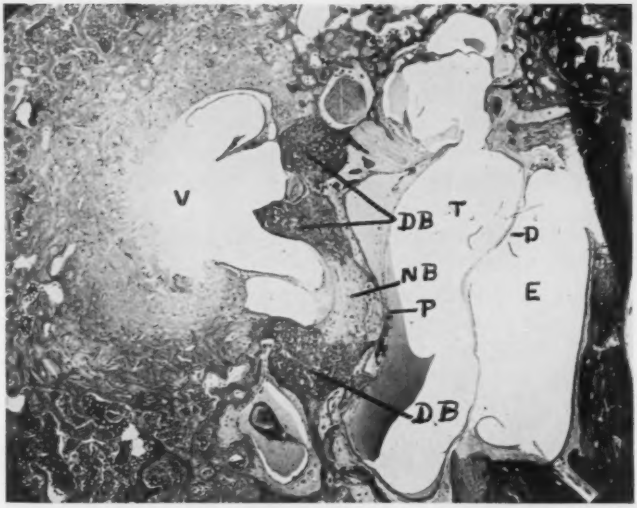
Same description as Slide No. 10.

Here we see an invasion of the tympanic cavity by the cholesteatomatous mass.

Slide 12. Suppuration of Antrum and Mastoid.

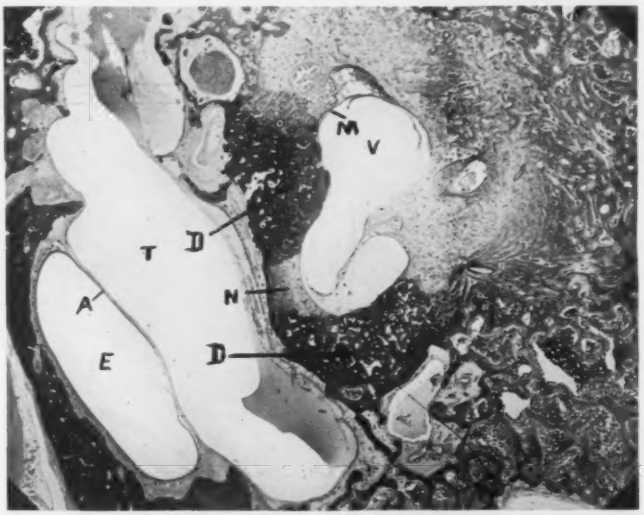
We see in this picture a large perforation of drum, and a mass of partially organized tissue in the middle, together with a diffuse round cell infiltration.

We see the aqueductus Fallopii and its contained nerve, and directly behind it we see the horizontal canal both on the inner wall of the antrum, leading off from the nerve we see the stapedius muscle. The antrum and a large excavated cavity back of it is filled



No. 21. Otosclerosis.

Slide 21. E. External auditory canal. D. Drum membrane. T. Tympanic cavity. P. Promontory. V. Vestibule. D.B. Diseased bone around oval window. N.B. Normal bone.



No. 22. Otosclerosis.

Slide 22. E. External auditory canal. A. Drum membrane. T. Tympanic cavity. V. Vestibule. M. Vestibular nerve. N. Normal bone. D.D. Diseased bone.

with a partially organized, partially purulent and necrotic mass, that it is partially organized is shown by the presence of young stellate connective tissue cells and by the presence of endothelial capillaries. This large cavity is due to necrosis of the septa between the cells. An interesting condition is seen in the extended growth of the epidermis on the outside of the drum through the perforation and into the cellular exudate which totally occupies the tympanic cavity.

This extension of epidermis is unaccompanied by the fibrous layers and the mucous membrane layer.

Gleason states pathologically and surgically the structure of the mastoid process is of the utmost importance. In the pneumatic type, pus from the antrum readily finds its way to the lateral surface. but in the diploetic, and more especially in the sclerosed type, there is greater danger of pus burrowing its way into the cranial cavity.

Slide 13. Diphtheria of Eustachian Tube.

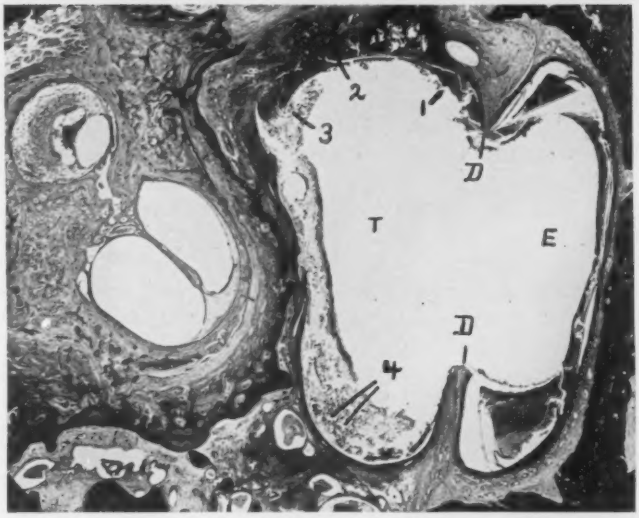
Section through the bony canal.

This is a cross-section view of Eustachian tube of a patient that died of diphtheria.

Here we see the tensor tympani muscle on top, next we see the processes cochlearis or the bony shelf, and next is the Eustachian tube. The epithelial covering of the mucous membrane of the tube consists of extremely tall, stratified, ciliated, columnar cells on the floor of the tube. This is due to the fact that the secretion is propelled along the floor as it drops to the lowest point of the tube, as the epithelium extends upward to the roof of the tube it changes into low cuboidal, apparently nonciliated.

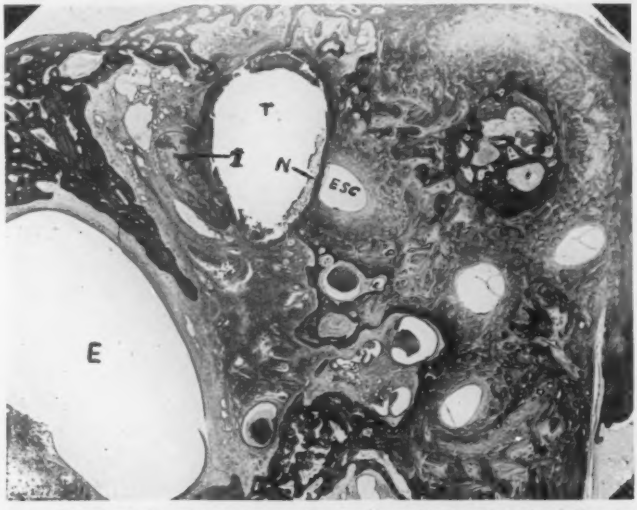
The tunica propria consists of dense, fibrous tissue, in which we see a great many mucous glands and blood vessels. Towards the top we see a muscularis mucosa, on one side papilla in the tunica propria are seen. The periosteum is pronounced. In the lumen of the tube we find a mass of all kinds of leucocytes, with a lot of loose, free black dots and looking on one side of the tube near the top we find a destruction of the epithelial layer and part of the tunica propria. No fibrin seen in the cell mass or in mucous membrane.

The toxin of the firm has completely dissolved and broken down the cytoplasm of the cells, but it did not seem to affect the nucleus as they are seen intact in great numbers. This mass is not organized. I don't think the dots are the nuclei of the leucocytes.



No. 23. Tuberculosis of the Tympanic Cavity with Perforation of the Drumhead.

Slide 23. E. External auditory canal. D. Stumps of drum membrane. T. Tympanic cavity. 1. Gathering of endothelial leucocytes. 2. Dense fibrous tissue. 3. Area of red cells. 4. Two tubercular giant cells.



No. 24. Tuberculosis of the Tympanic Cavity with Partial Necrosis of the Head of the Malleus.

Slide 24. E. External semicircular canal. T. Tympanic cavity. 1. Malleus. E.S.C. External semicircular canal. N. Necrosis of bony wall of external semicircular canal.

SLIDE 14. SCARLATINA OF EUSTACHIAN TUBE.

Section through the cartilaginous canal.

This is a cross-section of the Eustachian tube in which the patient died of scarlatina.

The section is through a portion of the tube in which the epithelium is stratified, ciliated columnar.

In the lumen of the canal we see a necrotic mass and outside this necrotic mass we find thick strands of deeply staining fibrin, with all kinds of leucocytes enmeshed and along this area the epithelium of the mucous membrane and a portion of the papillary layer of the tunica propria is destroyed and in the tunica propria we see empty blood vessels filled with fibrin, showing that this disease produces a lot of fibrin with subsequent damage as result.

Politzer states that: The most severe forms of acute suppuration of the middle ear develop very often in the course of scarlet fever more frequently, however, when complicated with nasopharyngeal diphtheria.

Primary diphtheria of the pharynx is seldom complicated with aural affections.

Two forms of diphtheria process in scarlet fever.

1. True diphtheria, with formation of diphtheria membrane upon which the Loeffler bacillus has been found, and later the staphylococcus, diplococcus, and the streptococcus, through secondary infection.

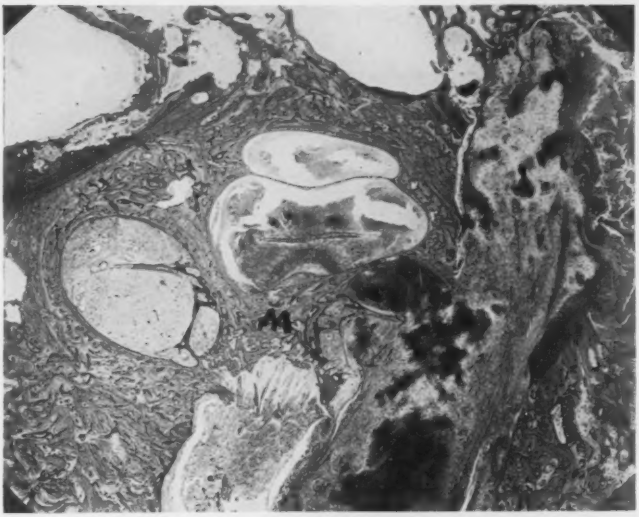
2. Necrotic scarlatinal-diphtheria, brought about by a marked streptococcic infection. It is in this later type that the destructive middle ear suppurations associated with swelling of the glands are developed.

The scarlatinal-diphtheritic middle ear suppuration generally develop at the crisis of a nasopharyngeal diphtheria which causes them, seldom during its decline.

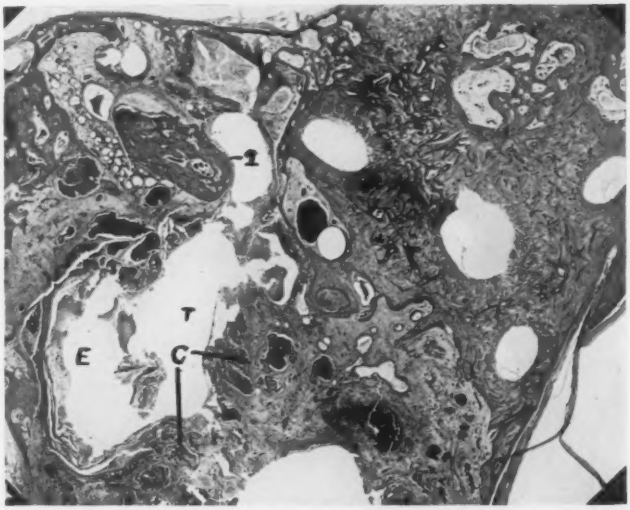
SLIDE 15. CHRONIC OTITIS MEDIA, WITH FISTULA OR EXTERNAL SEMICIRCULAR CANAL. DIFFUSE PURULENT LABYRINTHITIS.

This picture shows a large perforation of the drumhead with a turning in of the edges, and a partial ingrowth of the modified skin stratum. In the tympanic space we see an organized mass of connective tissue, with a generous sprinkling of dense fibrous tissue resembling the middle fibrous layer of the drum, from which it is most likely derived. This is hyaline in character.

Now this organized connective tissue is greatly infiltrated with all kinds of leucocytes; we see a great many capillaries. Here we see the bone forming the aqueductus Fallopii on the antral side missing



No. 25. Tubercular Caries of the Cochlea.
Slide 25. M. Modiolus.



No. 26. Carcinoma of Middle Ear.
Slide 26. E. External auditory canal. T. Tympanic cavity. 1. Malleus. C. Areas of carcinomatous mass.

and we see a greatly increased thickness of the fibrous tissue adjacent to the periosteum surrounding the nerve.

Posterior to the nerve we find part of the bone forming the horizontal semicircular canal on the antral side necrosed and missing, but leaving exposed and intact the membranous canal at its point of attachment to the concave side of the bony canal at the point.

Here again we find partially organized tissue greatly infiltrated with all kinds of leucocytes and also we find capillaries in this tissue adjacent to this area in the tympanic cavity. There has been an invasion of the purulent material through this hole in the wall of the bony canal into the perilymphatic space, but the endolymphatic space is free from invasion. The infection has spread through the entire canal system, but at no point does it show that the endolymphatic system has been invaded; nothing but the perilymphatic system.

This is the type of case that gives the fistula symptom.

This type of case seems to have produced a great deal of fibrous tissue, as seen in the middle layer of drum; also a great deal is seen in the corium of the external ear canal, below the epidermis.

As no tuberculous giant cells are seen, this case must have been an old chronic otitis, nontubercular in type.

Rutten states that nearly all the diffuse purulent labyrinthitis have their causes in chronic middle ear suppurations. Seldom do they occur in acute otitis.

Politzer gives as the three particular causes:

1. Cholesteatoma (Kümmel).

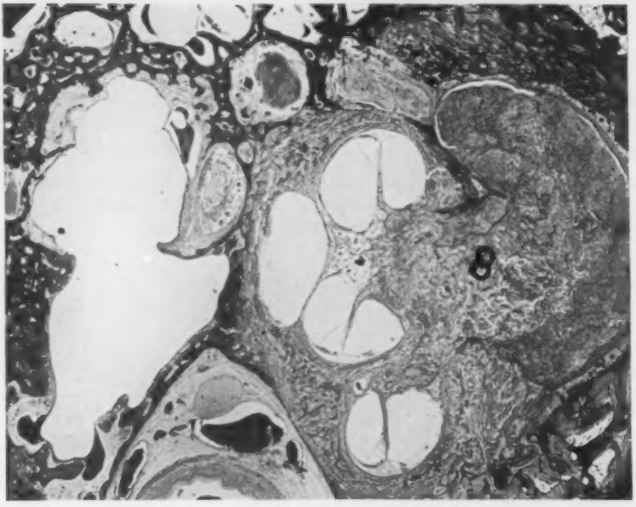
2. Tubercular middle ear suppurations.

3. Sepsis brought about by the pus retention in neglected cases, which lead to rupture of the labyrinthian capsule.

The places of rupture are: *1.* The oval window; *2.* the round window; *3.* fistula in the horizontal, less often in the posterior semicircular canal; *4.* fistulous perforations in the promontory wall.

SLIDE 16. CHRONIC OTITIS MEDIA WITH LABYRINTHITIS.

Here we find the drum perforated, and we see the epidermis growing over the edges into the tympanic cavity and on to a mass of partially organized tissue. The infection has spread in to the labyrinth and we find a mass of pus cells in the scala tympani at the secondary tympanic membrane of the round window, also in the mass in the tympanic cavity we see an accumulation of endothelial leucocytes in a great number of places practically fusing together.



No. 27. Metastatic Adenocarcinoma of Auditory, or Eighth Nerve.
Slide 27. 8. Eighth nerve.

No. 28. Dural Endothelioma of Eighth Nerve.
Slide 28. 8. Eighth nerve.

SLIDE 17. LABYRINTH SUPPURATION AND LATER ORGANIZATION OF THE SAME.

In this picture the drum is perforated and we have in the tympanic cavity organized tissue with many saccules.

The labyrinth has been inflamed and later young connective tissue grew in and obliterated the scala tympani, and there is an actual growth of new bone in the scala tympani and it extends up along the pectinate ligament. Corti's organ is thinned out.

SLIDE 18. ATROPHIC LABYRINTH.

This picture shows no evidence of recent inflammation. No purulent material present, nor subsequent new ingrowth into either the perilymphatic membranous system or the endolymphatic membranous system of connective tissue. Reissner's membrane is intact and normal.

The important thing to be seen is the thinned-out tectate membrane plastered down over an area that normally should be occupied by Corti's organ.

Corti's organ is a crumbled mass lying on the basilar membrane. An important thing I note is a break in the lamina spiralis ossea at its junction with the modiolus, though the spiral nerve is present in both parts at the areas of missing bone.

When I speak of the perilymphatic membranous system and the endolymphatic membranous system, I mean that an analysis of the labyrinthian capsule, from without inward, shows that there is:

First, a bony system of communicating cavities, the walls of which are bone. This is seen in a macerated specimen of the temporal bone in which all the tissue is removed, dried and sectioned.

Secondly, the walls of the entire bony system are lined by a layer of tissue that varies in places, and is the counterpart of the bony system. This is the perilymphatic system of membranous communicating cavities, the walls of which are composed of periosteum on the outside entirely at its attachment to the bony system, and endothelium lining the inside of the cavities. It varies at the basilar membrane.

Thirdly, located in the perilymphatic system of membranous communicating cavities and attached to its walls on the inside is the third system or the endolymphatic system of membranous communicating cavities, but not the counterpart of either the bony system or the perilymphatic membranous system.

The endolymphatic system is attached to the walls of the perilymphatic system along certain lines of various widths.

The walls of the endolymphatic system are composed of endothelial tissue on the perilymphatic side, and epithelial tissue on the endolymphatic side, except at the lines of attachment. At the lines of attachment of the endolymphatic system to the perilymphatic system, the walls consist of periosteum on the outside with the intervening connective tissue and epithelial tissue on the endolymphatic side.

The anatomical causes of congenital deafness are very numerous and I refer you to Politzer's book, p. 822.

SLIDE 19. MASTOIDITIS, LABYRINTHITIS, SUPPURATIVE MENINGITIS.

In this picture we have evidence of suppuration in the scala tympani, media and vestibuli. Corti's organ is normally present, but the tectate membrane is thinned out and plastered to it and fixed. Pus cells and leucocytes are found in the cavity and in vicinity of Corti's organ. The linea striae shows the blood vessels engorged and congested. In a study of labyrinthine affections we find that scarlatinal diphtheritic otitis causes a purulent labyrinthine inflammation more often than a primary otitis, and is the form that usually causes deaf mutism.

SLIDE 20. OTOSCLEROSIS.

This picture presents the following:

In the external ear canal the skin is greatly thickened. The epidermis is extremely thin with the stratum germination very pronounced. The corium is rather thick. The drum is present, but the various layers are thin. The tympanic cavity is extremely large, with little or no cellular exudation present. The most important thing is the change in the bony structure of the footplate of the stapes and the external wall of the vestibule surrounding the stapes for a short distance. We find the footplate of the stapes increased in thickness to ten times its normal size, also it is pushed into the vestibule on the inferior side. This change has taken place most likely all around the fenestra vestibule. A sharp line of demarcation is seen where the new bony formation meets with the old bone, the direction of the layers of the new bone is at right angles to the direction of the layer of the old bone. Osteoclasts are seen in spaces of the footplate. The vestibule shows no cellular exudate.

Politzer states that a typical case of otosclerosis is a primary affection and not dependent on disease of the mucous membrane. Stapes ankalosed if the oval window is involved, differentiated from the normal by the deep carmine stain, it can involve the entire labyrinthian capsule. The pathological changes show the newly formed bone, characterized by the immense size of its bony spaces and

Haversian canals. The osseous spaces are filled with connective tissue rich in cells, which surround large and small blood vessels. He has seen osteoblasts and osteoclasts around the blood vessels. Alexander says, that the cause of otosclerosis is unknown. If due to hereditary syphilis the positive Wassermann reaction as in syphilis should be more positive in a greater number of cases, but such is not the case.

Anatomy—In otosclerosis there are found pathological foci of bones, which in the shape of sharply demarcated tumors often replace the normal bone of the capsule. In the histological examination the normal bone stains bright red under application of hematoxylincosin, while the otosclerotic foci assume a reddish-blue or deep blue color. The otosclerotic foci have very large hollow spaces, which are permeated by pathologically enlarged blood vessels. There is a rather dense network of connective tissue spread out between the bone and the vessel walls. The favorite seat of these foci is the lateral wall of the labyrinth. He calls the condition osteitis vasculosa.

SLIDE 21. OTOSCLEROSIS.

This picture shows the areas of both the oval and round windows involved. No other pathological condition seen. Description same as Slide No. 20.

SLIDE 22. OTOSCLEROSIS.

Here we have an involvement of the labyrinthian wall. There are areas in which the change has taken place, and there are areas in which the change is taking place.

In this picture we see it advancing slowly and already involving the vestibular nerve.

SLIDE 23. TUBERCULOSIS OF THE TYMPANIC CAVITY WITH PERFORATION OF THE DRUMHEAD.

In this picture we see the drumhead with a large perforation. The stumps of the drum on both sides are thicker than normal. The thickness is due to an intense proliferation of the cells of the fibrous layer. The epidermis has grown over the edges and into the tympanic cavity. At the ending of the epidermis on the upper side is seen an accumulation of large endothelial cells, perhaps gathering to form a giant cell. What is seen in the tympanic cavity is nothing but a mass of dense fibrous tissue. This seems to come from the periosteum. Of course all newly organized tissue is evidenced by the presence of many congested blood vessels. Resting on this newly organized tissue is a layer of cellular exudate with a diapedesis

of red cells interspersed. In one area of the layer outside of the fibrous tissue is seen two tubercles with typical tuberculous giant cells in them. The appearance around Corti's organ is an artefact, as I don't see an infiltration of cells or exudate in the cochlea.

Maximow in his article, "Tuberculosis in Mammalian Tissue in Vitro," experimental aseptic inflammation, states, lymphoid tissue culture.

The development of the mononuclear ameboid inflammatory cells, the polyblasts are developed partly from the emigrated blood lymphocytes and partly from the mobilized resting wandering cells. These polyblasts engulf the tubercle bacilli, hypertrophy and transform themselves into typical epithelioid cells. The tubercle bacillus affects the fibroblasts very little. The reticular cells play the most important role in lymphoid tissue in cultures inoculated with tubercle bacilli. The reticular cells are phagocytic for tuberculous germs; and they undergo mitosis and also hypertrophy. Reticular cells are special fibroblasts. There is no evidence of the transformation of ordinary endothelial cells of the blood or lymph vessels being changed into wandering phagocytes or polyblasts.

The chief cause of the giant cell is from the reticular cells mobilizing as free elements and become changed into epithelioid cells. He could never find any indications of endothelial or fibroblastic origin of the giant cells. The principal mechanism of the giant cell formation is the fusion of mononuclear cells. This phenomenon is to be looked upon as a further step in the clustering of the cells and of tubercle formation under the influence of the chemical stimulus originating in the bacillus. It must be due to a sudden decrease of surface tension. The tuberculous giant cells may form without direct mechanical contact with the tuberculous bacillus and may not even contain the tubercle bacillus.

Caseous Degeneration—The fused mononuclear cells undergo a change into a caseous degeneration, hard to differentiate between ordinary coagulation necrosis.

Lymphocytes—The highly virulent bacilli do not multiply rapidly, and are soon engulfed by the cells forming the tubercles. The nonvirulent bacilli proliferate excessively. Therefore, the picture we see in a tuberculous lesion is an area of necrosis due to a fusion of the large mononuclear reticuloendothelial cells, which are dead and the receding nuclei at the periphery of the tubercle, a layer outside of this containing epithelioid cells and the layer beyond this is a layer of lymphocytes. Here we see it true to form.

SLIDE 24. TUBERCULOSIS OF THE TYMPANIC CAVITY WITH PARTIAL NECROSIS OF THE HEAD OF THE MALLEUS.

Tuberculosis of the tympanic cavity with partial necrosis of the incus and a necrosis of the bony wall of the external semicircular canal, but with the membranous canal intact, the middle ear cavity is filled with newly organized tissue. There is, as seen in the other slides, a tremendous amount of fibrous tissue. A great number of congested blood vessels are seen in this tissue, together with a great deal of diapedesis of red cells. The incus is partially necrosed. Throughout this cellular exudate is seen a great many giant cells. The bony horizontal semicircular canal is necrosed at the attachment of the membranous canal.

SLIDE 25. TUBERCULAR CARIES OF THE COCHLEA.

In this picture is seen great destruction of the labyrinth, not alone to the bone, but to the tremendous stimulation of the connective tissue into the cochlear tube to its total obliteration of all normal structures contained therein. The wall to the right of the modiolus is destroyed or necrosed, and the scala tympani and scala vestibuli are filled, together with an overgrowth of new connective tissue and a necrotic mass, whereas, on the left of the modiolus the bony canal is intact, but completely filled with organized tissue without necrosis. In the necrotic mass is seen numerous giant cells. The membrana basilaris is completely gone on both sides. The lamina spiralis ossea on the left is intact, while there is a leucocytic infiltration in the spiral or bipolar ganglia in the spiral or Rosenthal's canal. On the right the leucocytic infiltration is more pronounced. There is a leucocytic infiltration between the fibers of the eighth nerve. There is a tremendous amount of what looks like fibrous tissue throughout the lesion, but it may be a hyaline or fatty change before necrosis.

SLIDE 26. CARCINOMA OF MIDDLE EAR.

The drum is gone and in the tympanic cavity we see an invasion by the carcinomatous mass. Whether this originated in the external ear or the tympanic cavity or whether it is metastatic, I don't know; but analysis of the various structures show a highly vascularized, greatly thickened corium of the skin.

The tympanic cavity is practically filled with the new growth, and the incus, in two places, have been destroyed and replaced by the carcinomatous tissue. In carcinoma we have not alone the epidermis, but also the corium to deal with. The facial canal is extremely large and in the facial nerve there are two areas that

look like an invasion of it, but not through the bony wall at that point, as it is intact.

The external semicircular canal, like the aqueductus Fallopii, is extremely large, but this may be due to the plane of the cut, and the wall on one side is undergoing a change, in which the periosteum and endothelial layer is absent.

Alexander states that the majority of carcinomata of middle ear are due to chronic middle ear suppuration.

Politzer states that epithelial new formations develop most frequently on the auricle and in the external auditory canal, less frequently in the tympanic cavity and the mastoid process.

According to Mallory, a tumor is a new formation of cells which proliferate continuously and without control, tend to differentiate as the cells from which they arose, serve no useful function, lack an orderly structural arrangement, and have no cause for their existence.

Of the epithelioblastoma, the three most important are:

1. Papilloma (an epithelial tumor in which cells cover finger-like projections).

2. Adenoma (an epithelial tumor in which cells have gland-like depressions or cavities in the stroma).

3. Carcinoma (an epithelial tumor which infiltrates and which may give rise to metastasis).

Carcinoma, therefore, is an epithelial tumor which infiltrates and which may give rise to metastasis. It is the term applied to all epithelial tumors which pathologically invade and which clinically are malignant. Carcinoma may grow in any of the three ways described.

In dealing with epithelial tumors, the stroma must be taken into consideration. In an epithelial tumor which grows above a surface, the stroma is all new-formed.

Epithelial cells, both in the normal organs and in tumors arising from them, directly influence and determine the amount of the stroma. In other words, that epithelial cells, chemically or otherwise, can cause proliferation of fibroblasts.

Carcinoma is an epithelial new formation which tends to invade the lymph spaces of the connective tissue adjoining and gives rise to metastasis.

Slide 27. Metastatic Adenocarcinoma of Auditory, or Eighth Nerve.

In this picture is seen an involvement by metastasis of the eighth nerve. This growth has not only extended clear to the fundus of

the internal auditory canal, but has penetrated the canals in the modiolus, especially the spiral canal or canal of Rosenthal, and in this canal the tumor has eroded, I presume, by pressure toward the modiolus side, and has increased the size of it considerably.

The bipolar cells of the spiral ganglion cannot be distinguished, due to the presence of the tumor cells. The growth has extended into and between the anterior and posterior leaves of the lamina spiralis ossea, and also is seen an invasion of the pectinate ligament at one place. An adenocarcinoma is a carcinomatous tumor whose cells line gland-like depressions or cavities in the stroma.

SLIDE 28. DURAL ENDOTHELIOMA OF EIGHTH NERVE.

Again is seen a tumor which has extended to the fundus of the internal auditory canal.

It is an endothelioma, showing fibrous and spindle cell structure. There is an area from a line which most likely constitutes the fundus and also the limits of the tumor to another line further in the modiolus devoid of bone and along the inner line is seen evidence of erosion.

Dural endotheliomas are characterized by whorls.

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3330 Chestnut Street.

DISEASES OF THE NASAL ACCESSORY SINUSES COEXISTING WITH INTRACRANIAL CONDITIONS: CASE REPORTS.*

DR. A. LOBELL, New York.

It is customary to assume that the various signs and symptoms which a patient presents are caused by a certain disease. While this system works satisfactorily in most instances, nevertheless, there are occasions where grave errors are the result of this.

In the present day of specialization, the possibilities for such mistakes are abundant because each specialist becomes profoundly interested in his or her limited sphere, thus bringing about a narrow point of view. Incidently, it might be mentioned that this is one of the arguments that should be advanced in favor of adequate training in general medicine before specializing.

While many branches of medicine and surgery overlap each other in their scope, none are so closely related as the branches of ophthalmology, rhino-otology and neurology.

Owing to the anatomic relationship between the nasal accessory sinuses and intracranial structures, the rhinologist must be on the alert for intracranial complications and be able to recognize them. In doing this, he must not overlook the possibilities of intracranial neoplasms and inflammatory processes of independent origin. Such conditions may coexist in a patient who is under the care for sinusitis and may easily escape detection if precautions are not taken. To illustrate the practical significance of the above and the importance of consulting the neurologist in all such instances, the following cases are reported.

Case 1: Ethmoiditis Coexisting with Encephalitis Lethargica. M. W., male, age 38 years, married, manufacturer.

Family History: Two brothers and one sister suffered from chronic purulent sinusitis.

Past History: He had many attacks of acute sinusitis. He was subject to frequent colds. He had no injury to his head and he never had syphilis.

Present History: On June 9, 1924, he noticed that he was unable to open his left eye. Lifting the upper lid with his fingers, he was convinced that his sight was undisturbed. He consulted an ophthalmologist and he was informed that his condition was due to some focal infection within the sinuses or teeth. On June 14 he was referred to the writer for consultation.

*Read in part at the Lebanon Hospital Alumni Asso., March 12, 1926.

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Examination: The left eyelid was completely ptosed (Fig. 1). A neurological examination revealed that the left elevator palpebrarum superiorus, which is supplied by the third cranial nerve, was the only muscle involved. The temperature, pulse and blood pressure were normal. A urinalysis was negative. The blood Wassermann was negative and the cerebrospinal Wassermann was not determined because the patient refused a lumbar puncture. A rhinoscopic examination suggested the presence of ethmoiditis. Roentgenograms of the sinuses and teeth showed a moderately severe involvement of both ethmoidal regions and apical infection of four teeth. The tonsils were apparently normal.

Figure 1.

Figure 2.

Fig. 1. Complete left ptosis: occipito-frontalis relaxed.
Fig. 2. Complete left ptosis: occipto-frontalis contracted, raising the lid, partially. Note left eyebrow raised to a higher level than the right.

Treatment: The teeth were extracted and the patient was under the impression that he was improving. However, I was convinced that the improvement was imaginary, being due to forcible contraction of the occipito-frontalis muscle, thus lifting the lid by raising the forehead (Fig. 2). As a therapeutic test for a possible gumma, iodids were administered until the patient showed signs of iodism, but there was no improvement. The next logical step was the consideration of the exenteration of the ethmoids. Fortunately the operation was postponed.

In the beginning of September, 1924, he developed definite signs of encephalitis lethargica, from which he eventually recovered.

Comments: This case clearly demonstrates how important it is to think of encephalitis wherever there is an involvement of a cranial nerve without a demonstrable cause. It was merely accidental that the patient escaped an operation on the ethmoids. The operation might have aggravated the encephalitis or reduced his chance of recovery by diminishing his vitality and resistance. Had he submitted to a lumbar puncture, a correct diagnosis might have been made earlier.

Case 2: Maxillary Sinusitis Coexisting with Encephalitis Lethargica.

History: F. H., female, age 14 years. In the latter part of September, 1923, she suffered from an attack of grippe which lasted four days. There were no sequela, except that she had a mucopurulent discharge from the nose. On Oct. 6, she began to complain of lancinating and darting pain which started in the left superior maxillary region and radiated toward the left ear, mastoid, neck, shoulder and arm. She was restless and had a total uncontrollable insomnia. Internal medication and various local applications were all in vain. The writer was called to see her Oct. 12, 1923.

Examination: She appeared acutely ill and in severe pain, pulling the skin of the painful areas, tossing about and crying. The temperature was 100° F., pulse 110. A rhinoscopic examination showed the presence of mucopurulent material in both middle meati and the mucous membrane was uniformly congested. The left lower lid was slightly edematous and the left maxillary region was tender. The ears and mastoids were normal.

Since the patient was confined to bed, it was inconvenient to transilluminate or X-ray the sinuses. It was decided to puncture and irrigate the left antrum for a diagnostic purpose. The return flow contained small lumps of mucopurulent material, the liquid was blood-tinged and cloudy. However, this did not relieve her pain. The left sphenopalatine ganglion region was cocainized in order to relieve the pain, but there was no relief. It was therefore evident that her pain did not originate peripherally and an intracranial condition was thereupon suspected. Furthermore, the restlessness and absolute insomnia spoke in favor of a cortical involvement.

The patient was then examined by a neurologist. Subsequent developments proved the condition to have been encephalitis lethargica. She recovered after a prolonged illness and convalescence.

Comments: From the very outset it was apparent that a maxillary sinusitis even of the severest type could not account for the restlessness, insomnia and prostration. The writer is not aware whether the cocainization of the sphenopalatine ganglion region has been de-

scribed as a differential diagnostic test between central and peripheral pain in this region. At any rate it proved of great value in this case, because it gave the real clue to the situation.

Case 3: Sinusitis Coexisting with a Tumor of the Cerebellum. M. S., male, age 24 years, single, painter, presented himself for examination on Nov. 12, 1923.

History: For three months prior to this date, he suffered from paroxysmal attacks of headache. It was limited to the supraorbital and frontal regions, aggravated by stooping, and it recurred at irregular intervals, mostly in the afternoons. He also had a mucopurulent nasal discharge and was subject to frequent colds. He had already consulted other nose and throat specialists, but they failed to relieve his headache. He never had syphilis.

Examination: A healthy looking man, well developed and nourished. There was tenderness over both frontal and supraorbital regions. Both nasal fossae contained many odorless crusts. The mucous membrane was moderately atrophied and the middle turbinates were impinging on the adjacent structures. Suction, transillumination and Roentgenograms showed the presence of a chronic frontal and ethmoidal sinusitis. Conservative treatment caused a diminution of the discharge, but the headache persisted. A blood Wassermann was negative.

Course: On Nov. 28 the headache was spreading to the parietal regions and he vomited several times. He was dizzy but he had no distinct vertigo. The vomiting was projectile in character and had no relation to meals. The pulse was 52, full and bounding. The ocular fundi were normal. There was no spontaneous nystagmus or ataxia.

This attack, naturally, pointed to an intracranial condition. In as much as there were no signs of cerebral localization of any lesion, and since he had a chronic frontal sinusitis, it was thought he might have been suffering from an abscess in the silent area, namely, the frontal lobe.

The family was advised of the gravity of the condition and he was ordered to a hospital. That same night he died, suddenly, immediately following a lumbar puncture. An autopsy showed a large glioma of the vermiform lobe of the cerebellum. In the opinion of the pathologist, this tumor must have been there more than a year, because the ventricles were uniformly distended.

Comments: This case illustrates how careful one must be in dealing with persistent headache in the treatment of sinusitis. A diminution of the discharge calls for a corresponding improvement of the headache, provided the channels for drainage are not occluded.

It is of interest to note how a slowly growing tumor increases the intracranial pressure so insiduously that the patient has no complaints for a long period. For the same reason, presumably, there were no pathologic changes in the ocular fundi. In this patient the headache was definitely limited to the frontal area until the final stage. In tumors of the cerebellum, the headache is usually diffuse or occipital. This case again calls our attention to the danger of performing a lumbar puncture in cerebellar tumor or abscess. This patient died as a result of the withdrawal of the cerebrospinal fluid. The sudden release of the pressure caused the aspiration of the cerebellum and the tumor. The pressure exerted on the vital centers of the medulla caused immediate cessation of respiration and circulation.

REPORT OF CASE WITH AN UNUSUAL CONDITION INVOLVING THE NASAL SEPTUM.

DR. E. S. HALLINGER, Camden, N. J.

The presentation of this case is simply for the purpose of recording the peculiar, and I believe heretofore unrecorded findings, that were discovered on performing a submucous operation for the relief of an almost complete obstruction of the left nasal fossae, incidentally to alleviate periodic habitual headaches. The history of the case has no bearing on the subject-matter to be presented, yet revealed many points that are of interest: The record of the findings on the preliminary examination of the patient likewise can be omitted, with the exception that it may be mentioned, that no nasal or other operation had ever been performed, and that examination by all available means failed to reveal or demonstrate the presence of any unusual condition, particularly in reference to the septum posteriorly, as seen by posterior rhinoscopy.

On Oct. 14, 1925, at the West Jersey Homeo. Hospital, Camden, N. J., following the usual routine of preparation, and anesthesia, a vertical incision was made in the left anterior fossae, through mucous membrane and perichondrium to the septal cartilage; the muco-

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perichondrium was readily separated in all directions, after which the cartilage was incised to the perichondrium and mucous membrane of the opposite side, and likewise freed and elevated in all directions. A large quadrilateral segment of the cartilage was then removed by swivel knife, and on its removal it was observed that the posterior and superior border was unduly thickened; and on removing the remaining portion of the cartilage from its attachment to the vomar and perpendicular plate of the ethmoid, it was found to be almost ¼ inch in thickness.

This thickening became progressively greater above and posteriorly, and to a lesser extent below, approaching the normal, as was discovered on continuing removal of the deflected portion of the vomar; but the most pronounced change was that which was observed as occurring in the perpendicular plate of the ethmoid; here the bone was found to have a sharply defined *cortex* with intercortical bone of the soft spongy, cellular type, which was rather coarse in character, the interstices between trabeculi being rather large.

On continuing exenteration upwards, which was accomplished with some difficulty, as the mouth of the forceps would not grasp the area to be removed because the bony growth was too thick to permit forceps to embrace same, a large cavity was suddenly encountered; this was found high up in the perpendicular plate of the ethmoid, and on examination, with soft probe, was found to be approximately at least ½ inch in length, i. e., antero-posteriorly, and a full ¼ inch in its widest diameter. This cavity was found to be free of secretions, was smooth and glistening, and did not appear to be lined with mucous membrane or have communication with any other part of the nose. Its color was pearly grey, and on careful inspection as to its anatomical relations, both intra and extranasally, the roof of this cavity or cell was accepted as being part of the cribriform plate of the ethmoid. The thickening of the perpendicular plate of the ethmoid extended anteroposteriorly for some distance beyond the cavity encountered and gradually tapered off to the normal.

The operation was completed, the lateral walls of this cell being removed, together with all of the remaining deflected areas. The remnant of the septal cartilage was then lifted out of its bed along the maxillary suture line and the bony spur removed by means of chiselling; flaps were approximated and a large polypoid mass which was present in the right middle meatus was next removed. On inspection the right middle turbinate was found to be absent, the polypoid mass being attached to the hiatus high up, and to an open

ethmoid cell or depression posteriorly to the same. This polyp was multilobular and was 1 inch in its greatest diameter and almost ½ inch in thickness. The superior turbinate was very small. The left middle turbinate was found to be normal, and a few polypi were removed from the left middle meatus.

As mentioned previously, this case is being reported because of the unusual anomalous anatomical findings, which briefly restated are:

1. The presence of a large cell or cavity in the perpendicular plate of the ethmoid, the roof of same forming a part of the cribriform plate.

2. The character of bone making up or forming the perpendicular plate of the ethmoid, and to a minor extent, a part of the vomar, having the same or similar construction, or formation, as found in long bones.

3. The absence of the right middle turbinate, and the depression or cell from which grew a large multilobular polypoid mass; this absence being confirmed following the operation by a series of X-ray pictures, which were interpreted by two Roentgenologists of repute.

These unusual conditions as found are of interest, not only from a mechanical or operative viewpoint, but also because of the rarity of their occurrence, as I have as yet been unable to find mention of similar findings in the literature to date. In closing, the absence of the turbinate with the presence of a closed cavity or ethmoid cell in the perpendicular plate of the ethmoid, would seem to point to some relationship one with the other, probably a transposition.

517 Cooper Street.

CHOLESTEATOMA; CASE REPORTS.*

Dr. G. B. Trible and Dr. A. P. Tibbets, Washington, D. C.

The following cases of cholesteatoma are illustrative of the usual case, and of the extremely unusual, with extensive involvement; infection of the meninges and death.

Case 1: Female, Mrs. G. H. Fairly well developed woman about 40 years of age; family history negative so far as the ear condition was concerned; previous history negative, except scarlet fever at about the age of 12 years, complicated by a middle ear abscess which never cleared up, but kept up an intermittent suppuration. For the past two or three years had been very dizzy, was unable to walk or stand and had been troubled by an unusually bad odor in the ear discharge. On examination, it was found the right ear showed a purulent discharge, drum gone. Just in front, that is, towards external auditory meatus there was a small point of pus. Hearing in right ear *nil*, vestibular tests showed functioning labyrinth, radiograph report as follows:

"The right mastoid is composed of very small compact cells in its lower half. About this no cell walls are seen, the mastoid having a smooth, homogenous appearance. The sigmoid sinus is more prominent than on the left. Stereoscopic examination showed the left mastoid to be of the large cell pneumatic type, with no evidence of abnormality."

Small grayish particles from the discharge showed the characteristic cholestrin crytals and a diagnosis of cholesteatoma was tentatively made. On Jan. 28, 1924, a radical mastoid in the right ear was performed and a cholesteatoma mass about the size of a filbert was found hollowing out the upper part of the mastoid and extending to the dura, but separated from it by a thin, smooth, bony wall. Very little chiseling was needed, the mastoidectomy had been automatically performed by the rarefaction and absorption of bone by the encroaching mass. This case made an uneventful recovery, a later reinfection occurred from the throat, but subsided after a tonsillectomy. The radiograph report of a homogenous appearance in the upper half is thus explained; its was the cholesteatoma.

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*Read in part before the George Washington University Hospital Staff meeting by Dr. A. P. Tibbets.

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Case 2: Miss A., white, age 26 years, clerk. First seen by Dr. Tibbets as consultant with Dr. C. B. Conklin, her family physician, on May 13, 1924. Family history negative so far as her present condition was concerned; born and raised in the rural districts of Missouri, her mother and father being at that time remarkably well. Previous history negative in childhood, no ear trouble; the past October developed typhoid, returned to her home in Missouri for convalescence. Had complained at that time of a throbbing pain in the left ear at intervals for about a month. She had not consulted a specialist. Pain was not severe enough to keep her awake at night, nor to interfere with her daily routine. After her return to Washington, she at intervals complained of headache on the left side, but had not considered it of sufficient gravity to consult her physician. Present history and condition: Two days before developed an earache of moderate intensity on the left side, for which she had consulted Dr. Conklin, who found upon examination a slight redness along the handle of the malleus, but no bulging of the drum. Hot boric acid irrigations caused this pain to subside and she was perfectly comfortable until the next night at about 8 o'clock, when the pain returned with increased severity. Dr. Tibbets was called in consultation the next morning. Patient seemed in great distress, tossing and moaning of the pain in her head and apparently unable to answer questions coherently. Examination of the ear at that time showed decided bulging of the left drum, which was freely incised under primary ether anesthesia. A small amount of bloody serum followed the incision, but little relief was afforded the patient and her condition became so alarming that her friends were persuaded to transfer her to the George Washington University Hospital. A second more free incision was made that night, extending to the upper limit of the drum. The patient continued restless, delirious and had to be restrained. The following morning, May 14, marked signs of meningeal irritation were noted. A lumbar puncture was performed by Dr. Conklin, fluid found under pressure, 920 cells were found to a c.m.m., 97 per cent polys. Smears from the fluid were negative for bacteria. Stiffness of the neck, a positive Kernig and Babinski, and other signs of meningitis were present and the case was considered by the pathologist, Dr. Oscar B. Hunter, as one of pneumococcic or streptococcic meningitis. Dr. G. B. Trible was called in consultation, operative interference deferred until results of blood and spinal fluid culture which had been taken were reported upon. May 15, she developed a spastic condition in her right arm and involvement of the speech center, so that she was unable to pronounce words distinctly. The

cell count in the spinal fluid had gone up to 1580 and the culture was reported streptococcus hemolyticus. Culture from the ear discharge gave a mixed growth, staphylococcus pyogenes aureus and streptococcus hemolyticus. The blood culture was negative, the patient had to be restrained constantly and an X-ray picture could not be taken. The patient was seen again in consultation by Dr. Trible and decision was reached to explore the mastoid and the area above it in the hope that a localized area could be cleared out and pressure relieved at any rate. Upon exploration that evening, the mastoid was found to be of the small cellular type, rather thick cortex, very slight destruction of the septa and with but a few cells containing pus. Exploration of the roof of the mastoid antrum anteriorly showed necrotic bone and its removal was followed by the escape of about 2 drams of rather thick pus. All visible necrotic bone was removed and some soft substance, believed at the time to be necrotic brain tissue mixed with pus, a rubber drain was inserted, the wound partly closed. The patient showed considerable improvement following the operation, became quieter, began taking liquid nourishment through a tube and apparently recognized her father and mother, who had come from the West. There was evident interference with the speech center, for she would smile when spoken to, but not answer. A culture from the mastoid at the time of operation showed hemolytic streptococci in pure culture. Such marked improvement had been made that three days later, it was decided to establish freer drainage. The remaining roof of the antrum, the aditus and tympanic cavity were removed, the temporal plate above this and the middle fossa freely exposed and partly removed and a mass of whitish, odorless material resembling closely-packed macerated fish scales and extending over an area of about 2½ inches in length, 1½ or 2 inches in height, and of a depth of 1 inch at its thickest portion, removed, showing the dura intact beneath it, but the brain pushed over towards the right and apparently not involved, except at the posterior portion of this mass just above the mastoid antrum there was an opening where communication had been established and where the infection had evidently entered. Immediate laboratory examination of this material showed the characteristic cholestrin crystals. The patient reacted well from the operation and showed further improvement, the cell count in the spinal fluid went up to 17,000, practically all polymorphonuclears, with many of the cells in clumps, but negative for bacteria. On May 20, the wound was again reopened, small particles of cholesteatoma material was curetted out until the dura was freely exposed and clean. This had not been done before for fear of releas-

ing pressure too abruptly and time had been allowed for the brain to accustom itself to its expansion. The patient showed decided improvement, physically and mentally, became quieter and regained the use of the right arm and leg. She improved until the evening of May 24, when she began to fail, rallying somewhat the next day, but on the 26th became comatose and died at 6. p. m.

Comment: These two cases are widely different in their symptomatology and illustrate the two types of cholesteatoma unless we consider in this connection the true primary cholesteatoma without any history of previous ear disease and possibly arising from embryonic rests. Politzer, the great authority in otology, gives the usual origin of cholesteatomatous formations as a pushing in of the epithelium from the external auditory canal through the perforation and its continuation pathologically so that masses are broken off and tend to pursue independent growth, while other cases present the form and structure of cholesteatoma in other organs and may not begin to develop until after the suppuration has ceased. Virchow and others considered the presence of cholesteatoma in the temporal bone as a heteroplastic formation. Alexander also differentiates these conditions into primary and secondary, though he states the primary is scarcely ever encountered. It is occasioned, according to his theory by displaced embryonic epidermal cells, which grow independently. Secondary cholesteatoma follow the middle ear suppurations of a chronic type, epithelial cells penetrate the middle ear spaces through the perforation, a pathologic proliferation of isolated masses broken off or buried by suppuration, may continue until large masses are formed. McKenzie quotes and follows Politzer in this respect. Packard is of the opinion in cases where a small fistula is found in the labyrinth or in the petrous portion or isolated masses deep anywhere in the mastoid, the origin was not from the epithelium of the external auditory canal.

Unless removed the end results are infection of the mass, its breaking down, the production of a fetid discharge, softening of the already diseased surrounding bone and an extension into the meninges or sigmoid sinus, and death from meningitis or septicemia. In the second case given above, it is probable that the healed suppuration gave the necessary conditions for the beginning of the epithelial implant which probably went directly through a fissure in the antrum or attic, and the growth continued unrestricted by pressure until it attained its phenomenal size. This seems borne out by the finding of a collection of pus just above the roof of the antrum which probably originated from the infective agent in the second middle ear infection extending along the pathway of the epithelial

growth. So far as could be ascertained, no definite mental symptoms had occurred during a number of months this condition had existed. It is remarkable that such a large growth could exist in the middle fossa for such a time and cause no symptoms, for this is not a silent area. The shape of these masses is usually round or ovoid; the shape here was determined by the lack of restriction anteriorly and internally, for it pushed the dura away from a greater portion of the inner surface of the squamous portion of the temporal bone. There was no definite history of an actual perforation of the drum, the origin of the epithelial growth can only be surmised.

A SIMPLE, INEXPENSIVE SUBSTITUTE FOR THE "ARGYROL PACK."

Dr. J. B. H. Waring, Blanchester, Ohio.

In a considerable number of nasal conditions, where free depletion of the tissues is desirable, the time-honored "argyrol pack" is very efficacious. The treatment is quite mussy, as it were, and somewhat objectionable to our fastidious lady patients on that account; even though the treatment brings much relief to them.

A very effective substitute solution is a saturated solution of magnesium sulphate, "salts" in glycerin. This is colorless, and does away with the chief objection to argyrol; it is also far less expensive than argyrol and its many present-day modificatoins. Another advantage is that the solution keeps better than the various silver salts; and is ready for use over a considerable period of time, provided it is kept in a corked bottle. Owing to its deliquescent properties, its efficacy is lowered if left freely exposed to the air for any length of time. It may be argued that this substitute lacks the antiseptic properties of argyrol; this is true, but it is also a question as to how much antiseptic power is exerted by the argyrol in the average argyrol pack, taking its effect on the nasopharynx and sinuses as a whole. This same solution will be found of much value as a dressing for sprains, bruises and other superficial congestions and inflammations.

109 East Main Street.

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LATERAL CERVICAL FISTULAE.*

Dr. Rudolph Kramer, New York City.

Branchial or lateral cervical fistulae are well known, frequently discussed and seldom seen. The recent articles dealing with these fistulae are not in accord as to their embryological derivation, and their etiology and their natural history are indefinite because of the comparatively small number of cases observed.

Until 1913, when Wenglowsky published, in the Archiv. fur Klinische Chirurgie, his painstaking, accurate investigations of the embryological basis of lateral cervical fistulae, these fistulae were believed to be due to a failure of closure of the branchial furrows. According to some investigators the second branchial furrow was always at fault; according to others, the second, third or fourth might be involved. Wenglowsky clearly demonstrated that the thymic duct was the structure from which the lateral cervical fistulae originated. The thymic duct arises in the third pharyngeal pouch and passes downwards, outwards, and somewhat backward, then going off at an angle, it courses downward, inwards and forwards to reach the infrathyroid region, above the cardiac area. This duct usually becomes obliterated about the sixth week of fetal life, the obliteration starting, as a rule, from above and passing downwards. Portions of the duct might, however, remain patent and later give rise to lateral cervical cysts. The whole tract may remain patent and give rise to the various types of fistulae; the external or internal incomplete or the complete. The external or cutaneous opening is on a line parallel to and overlying the anterior border of the sternomastoid muscle, usually between the hyoid bone and the sternoclavicular articulation. The internal opening is almost invariably on the posterior palatine arch or directly in front or behind it. The fistulae generally present areas of squamous and of cylindrical (ciliated or non-ciliated) epithelium. There is a well defined circular coat of connective tissue present, and numerous lymphocytes often collected in distinct nodules are to be found in the wall. Muscle fibres and mucous glands are often present; less frequently, cartilage and hair.

Lateral cervical fistulae may be present at birth or they may appear in later life. Most frequently they first become evident at the

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age of two or three years. Leegaard observed a family tendency in a number of cases, and he also found that often the only indication of a lateral fistulae was a slight depression in the skin, about 1 to 3 c.m. above the sternum on a line with the anterior border of the sternomastoid muscle. The assumption made as to the late appearance of lateral fistulae is that the tract becomes infected and then ruptures externally. The course of the infective process has never been observed, so far as we could learn.

The external orifice is usually small, 1 to 3 m.m. in diameter. Varying quantities of serous or purulent fluid may escape from this opening; this discharge may be the only symptom present, and may be so annoying that it causes the patient to seek surgical relief. The external orifice may close for a time, days, weeks, or even years. There may be a swelling on the side of the neck, due to retention of fluid, but this is infrequent.

The internal or pharyngeal orifice may also be the site of discharge, the patient not infrequently complaining of a disagreeable taste as the result of the presence of pus or decomposed food. Injection of fluids with a distinct taste or a definite contrast color has been employed to detect the presence of a complete fistula. The elevation of the external orifice on swallowing is considered by Leegaard evidence of the presence of a tract, patent or partially obliterated, attached to the pharyngeal wall. Attempts at probing the entire tract have rarely been successful.

A very few cases have been stated to have healed up spontaneously. A few fistulae have been obliterated by the injection of chemicals, such as trichloracetic acid. The majority of healed cases have been treated surgically; that is, dissection of the entire tract.

Most of the patients have not submitted to any treatment, because the symptoms were not very numerous or disturbing and the surgical procedure fairly extensive, somewhat deforming and often unsuccessful. A few cases are reported to have undergone carcinomatous degeneration.

B. P., female, age 29 years, had had numerous attacks of acute tonsillitis, for which a tonsillectomy under local anesthesia was performed two and one-half years ago (May, 1923). General physical examination before operation revealed nothing abnormal except the tonsils, which were enlarged, cryptic and buried. She was discharged from the hospital on the second day after operation, in good condition, except for the throat wounds. Instead of gradually healing, however, secretion on the right side of her throat continued to annoy her. At times she expectorated pus. About three weeks

after the operation, a tender swelling appeared above the right sternoclavicular joint. The swelling ruptured a few days later, allowing the escape of pus. Since that time fluid in varying amounts, generally scanty, has escaped from the cutaneous orifice, which was just cephalad to the right sternoclavicular joint and overlying the anterior border of the right sternomastoid muscle. She has, at times, an unpleasant taste, ascribed by her to pus coming from the right side of her throat. At times a tender, slightly painful swelling appears just above the external opening and persists for several days. At these times very little discharge escapes externally. The discharge is generally thin and yellowish; occasion-

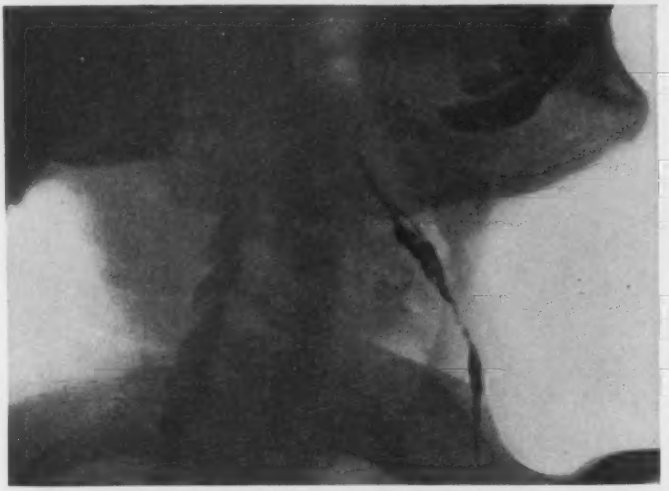

Fig. 1. Lateral view, showing fistula injected with bismuth and oil mixture.

ally a hair projects from the orifice and after being picked out, another takes its place at some later date.

When we saw her for the first time two years after her tonsillectomy, an opening about 3 m.m. long and 2 m.m. wide was seen in the right tonsillar fossa, anterior to the posterior pillar and below the mid-horizontal line of the fossa. A few drops of pus could be expressed from the opening. The margins were rounded and slightly elevated. The external orifice was situated 1 c.m. cephalad to the right clavicle and just over the anterior edge of the sternomastoid muscle. This opening was obliquely placed, 2 m.m. long and 1 m.m. wide, and leading into a tract directed upwards outwards

and slightly backwards. A drop of thin yellowish fluid was present in the orifice and a single, slender black hair protruded from it. About 2 c.m. above it, along the course of the anterior border of the sternomastoid, a small, firm, tender mass was felt. This mass was beneath the skin and not attached to it. A diagnosis of lateral cervical fistula was made and operation was advised. This advice was firmly refused by the patient. For this reason, tincture of iodin was injected in the attempt to obliterate the tract. This injection was preceded by cleansing irrigation of boric acid. At the first treatment no fluid passed from one opening to the other. One week later the external swelling had disappeared and on irrigating through

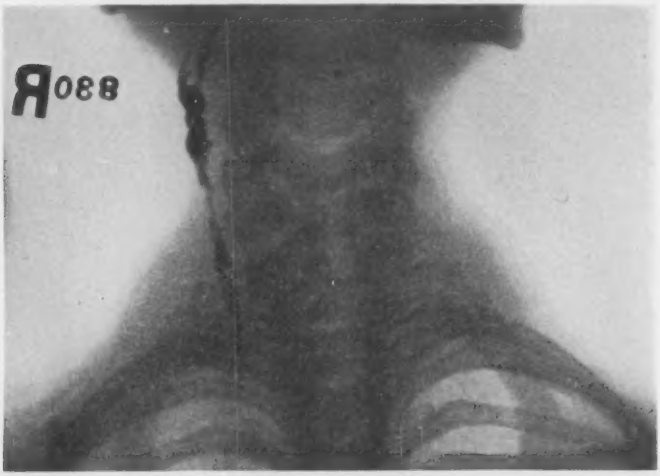

Fig. 2. Anteroposterior view showing injected fistula.

the internal orifice with acriviolet 1 per cent, a few drops escaped from the external opening.

Instillations were made at weekly intervals for a time and then we felt that an X-ray picture of the tract might be of interest. Accordingly, one probe was passed through the internal orifice and the fistula as far as possible, avoiding the use of force. The X-ray plates, taken by Dr. Arthur Bendick, show a gap between the ends of the probes of about 1 c.m. in the anteroposterior view and of 2 c.m. in the lateral view. A bismuth and oil mixture was then injected into the tract by means of a syringe and canula. The X-ray picture shows a continuous tract forming, near the upper extremity, an angle opening internally so that the upper portion of the

tract is directed somewhat laterally and downwards and the lower portion is directed mesially and downwards. There are several irregularities in the tract; one near the lower end corresponding to the region of the swelling of the neck; another near the angle of the tract, showing several indentations in its outline and an upward directed off-shoot.

Four days later the patient returned to the office with an abscess of the neck along the course of the fistula. As this assumed a serious aspect after a few days, the patient was finally prevailed upon to allow the incision and drainage of the abscess. (Operation by Dr. Leo Hahn, surgical service Dr. A. V. Moschcowitz, Mt. Sinai Hospital). The wound healed up in two weeks. The patient's condition is the same today as it was before operation, except for the scar of the external incision and the slight displacement downwards of the external orifice. Two hairs can be seen protruding from the cutaneous opening. The patient refuses to consider operative interference. Therefore, until she consents to operation we shall continue to irrigate the tract and perhaps to inject trichloracetic acid.

This case presents several interesting and unusual features.

First: The appearance of the fistula after tonsillectomy. We have not been able to find a case which presented a similar onset. It seems a valid assumption that the appearance of the fistula was due to the tonsillectomy. The internal orifice was laid bare and perhaps injured by the operation, and for several days after the operation it was bathed in pus and presumably occluded by membrane. The infection spread downwards along the tract to the lower end and here an abscess formed, just beneath the skin and then ruptured externally.

Second: The appearance of the subcutaneous tender swelling, associated with little or no discharge from the external orifice, and later its subsequent disappearance, with free passage of fluid from the pharyngeal to the cutaneous opening, is worthy of note. This obstruction of the tract and retention of secretions may be due to a sacculation which becomes filled and presses on the duct. The enlargement and irregularity of the fistula at this region, as shown by the Roentgen picture, tends to confirm this view. The retention is relieved after a time, perhaps by discharge into the pharynx and the lumen of the fistula is again patent.

Third: The presence of hair in the lower portion of the fistula is also unusual. Epidermoid structures, such as hair and sebaceous glands, have been found in some of the lateral cervical fistulae after

excision of the tract and minute examination, but the clinical appearance of hair growing from the fistula and presenting externally, is rare.

Fourth: The X-ray evidence is of interest in that it shows the course and the patency of the tract. This evidence is in strict agreement with Wenglowsky's reconstruction of the thymic duct, which he claims is the embryological forerunner of lateral cervical fistulae. We know of no case of lateral cervical fistulae demonstrated by radiography. Dr. Jay Whitham showed a case of bilateral lateral cervical (branchial) fistula at the October, 1925, meeting, which had been injected with bismuth and X-rayed. In searching the literature for similar cases, we found that in 1922, Lerche and Badolle published in the *Journal de Radiologie et Electrologie* two cases of median or thyroglossal fistulae injected with collargol and radiographed. It is unfortunate that an abscess followed the procedure in our case. We are inclined to believe that the infection resulted from an attempt to inject Beck's Paste. This was firmer than desirable for injection through the small canula used and perhaps the tract was injured in attempting to force the paste through the small lumen. The bismuth-oil injection which followed this attempt was easily performed. It would be advisable in these cases to use a mixture such as bismuth and oil rather than a mixture as viscid as Beck's Paste.

Fifth: Although a large abscess involved the lower portion of the tract and notwithstanding the fact that this abscess was incised and packed, there has been no demonstrable change in the patency of the lumen. It is for this reason that we have little faith in the efficacy of caustic applications to such fistulae.

A NASAL OINTMENT SYRINGE.

Dr. J. B. H. Waring, Blanchester, O.

In a considerable number of intranasal conditions, ointments of various composition are widely used by various physicians; some almost as a routine application, and others in special conditions. On the market are various ointment preparations, put up in collapsible tubes, and equipped with nozzles for either office or home application of ointment medication. These are satisfactory in a measure, so far as ointment application through the anterior nares is concerned superficially; but are practically useless when it is desired to apply ointment in sufficient quantity high up in the nose, or towards the nasopharynx; Eustachian orifices, etc.

The time-honored mode of procedure has been to apply the desired ointment on cotton-wound nasal applicators, and thus introduce into the nose; most of this ointment is brushed off along the septum or turbinates; and reaches its ultimate destination in minimal quantity, if at all. Not only this inefficiency of application, but the procedure is not unduly comfortable to any nasal passages; and is sometimes a cause of pain, where the nasal passages are congested and inflamed to start with.

To obviate this unsatisfactory and inefficient procedure in nasal treatments, the author has developed what may be called a nasal ointment syringe; that is, this was the prime object in view; but with appropriate nozzles the ointment syringe is well-nigh universal in its application of ointment to any desired body orifices, such as the ear, urethra, uterus, rectum, etc.

An all-metal syringe of standard type has been taken as a base; and with a few modifications, fashioned into a very satisfactory ointment syringe. It is impossible to force ointments through these small calibre nozzles by hand pressure on the ordinary syringe pistons; consequently this has been abandoned, and in its place a screw-thread piston has been substituted; and in place of the ordinary ringed end of piston, a stout T-shaped end has been made, to afford sufficient leverage for hand control. For nasal and aural use, a nozzle has been employed of fine silver tubing, very flexible; and bulbous-tipped at the distal end. This screws into the syringe proper by a standard thread; and hence is interchangeable with a number of other standard type tips. This flexible silver syringe tip can be fashioned into whatever shape the individual operator desires.

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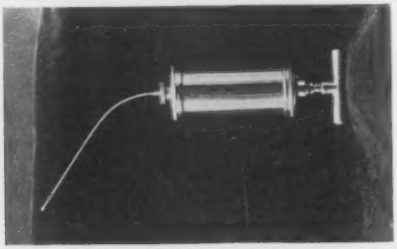
In use the syringe is filled with whatever ointment the physician desires to use; and the piston screwed down until a fine stream of ointment commences to issue from the end; the piston is then turned back a half-turn, which releases the pressure and stops the ointment flow; it is now ready for use.

A self-retaining nasal speculum is first placed in whichever side of the nose desired for treatment; after which the syringe nozzle is introduced into the nasal cavity between the septum and turbinates; the barrel of the syringe is grasped with one hand, usually the right; while the left hand is free to manipulate the piston control. A slight turn of the piston handle starts ointment slowly issuing from nozzle end in a steady stream, with which the end of nozzle is now carried under direct inspection to the exact portion of the nose it is desired to apply the ointment. By holding the tip steady at this spot for a few seconds, sufficient ointment quickly issues from the



Waring nasal ointment syringe.

syringe and is lodged as desired; after which the syringe nozzle is withdrawn from the nasal cavity, at the same time the piston handle being given a half-turn back to check ointment flow. The procedure is carried out expeditiously, and with a minimum of discomfort to the patient. Much less ointment is employed, as with the old method of application a good bit of it never reached the desired site, being wiped off en route.

The writer has employed this device for some time in nasal work; and derived much satisfaction therefrom; not only from an efficiency standpoint, but in added comfort to the patient. While primarily intended for the rhinologist, the additional nozzles available make the device of use to the general physician and surgeon as well. Manufacture is by an Eastern house.

109 East Main Street.

AN INSTRUMENT TO FACILITATE EUSTACHIAN TUBE CATHETERIZATION AND TREATMENT.

Dr. Louis K. Pitman, New York City.

The present method of treating middle ear diseases is inadequate in certain instances. For example, a case of acute otitis media is discovered at an early stage. The physician waits for the third day of the disease before making a paracentesis and following with the usual course of treatment. But he will have taken care only of the middle ear without taking into account the origin or path of the infection—the Eustachian tube. Nature may come to his aid and cure the Eustachian tube for him. On the other hand, serious complications may result, viz., such as chronic otitis, chiefly with the Eustachian tube perforation; retracted drum; anchoring of the drum; brain abscess; sinus thrombosis; etc.

It seems logical that the chances for these complications from middle ear infection would be reduced to a minimum by treating not only the middle ear, but the Eustachian tube as well. As a matter of fact, if the Eustachian tube were treated at an incipient stage of middle ear infection, the latter might be checked then and there. It is not the purpose of the author to report here on results of experiments with this new method, but

The Eustachian tube has been left alone in routine work in the past, because it has been impracticable to introduce a catheter into the middle ear through this tube. However, this difficulty is overcome by means of the instrument described below. With this instrument, which he calls a catheterizing Eustachascope, the author has been able to reach the middle ear through the Eustachian tube in some 15 seconds.

The catheterizing Eustachascope is an adaptation of the cystoscope by means of which a catheter is easily introduced into the middle ear by direct vision. In the diagram of it shown below, Fig. "A" is a tube through which the catheter is guided. It is easily fitted on an ordinary electric nasopharyngoscope. It may be slid backwards and forwards and also rotated about the axis of the pharyngoscope, as seen in Fig. "B." At "x" it is curved from its axis in order to facilitate the directing of the catheter

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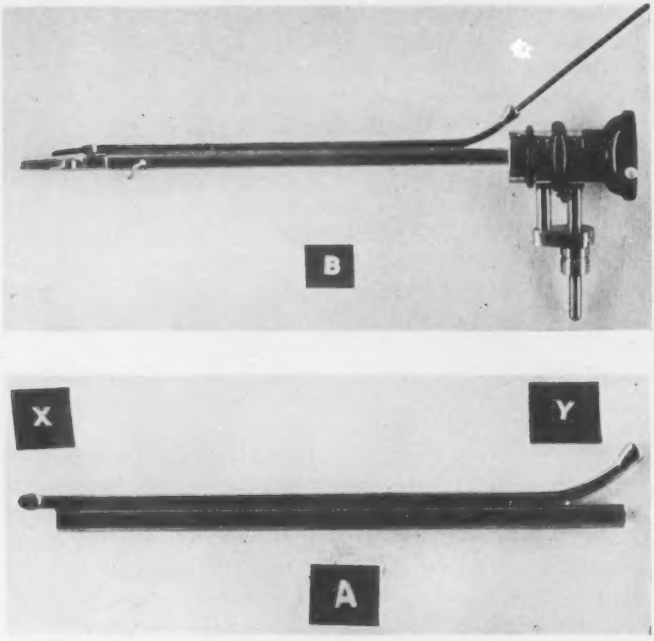
into the nasal orifice of the Eustachian tube. At "Y" is the opening for the catheter to slip it. Under slight cocainization the combined instrument is introduced into the nasopharynx in the same manner as an ordinary Eustachian catheter. The nasal orifice of the Eustachian tube is then brought into view and by manipulation the catheter is introduced into the canal.



The co-operation of Mr. Reinhold H. Wappler in the mechanical construction of the instrument is gratefully acknowledged.

The instrument is manufactured by the Wappler Elec. Co., New York City.

A subsequent paper will detail the results of treatment of middle ear diseases by this instrument and method.

simply to introduce an instrument for carrying it out.

221 West 112th Street.

COMMUNICATION.

THE LARYNGOSCOPE,
3858 Westminster Place, St. Louis, Mo.

Dear Editor:

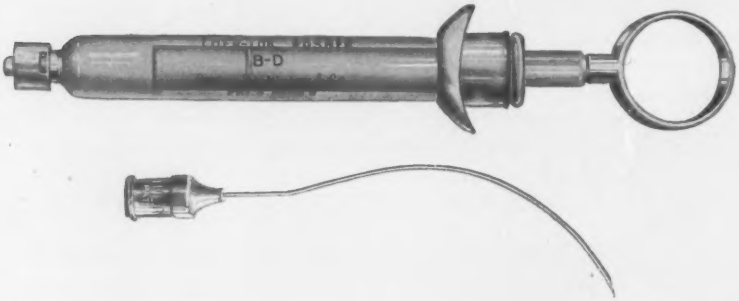
In the February, 1925, number of THE LARYNGOSCOPE, Dr. Simon L. Ruskin described his method of injecting the sphenopalatine ganglion by passing a needle up through the posterior palatine canal. In using this route, I found on several occasions that I could not get the needle through the canal, its point impinging on the posterior wall of the canal. This reminded me of Prof. Hajek saying, "Study the bones." Therefore, I examined all the skulls at my disposal and discovered that the canal is not straight, but conforms considerably to the curved surface of the maxillary bone which forms the anterior boundary of the sphenomaxillary fossa. This convex contour of the posterior wall of the antrum of Highmore is readily seen on looking at the skull. I then heated a long Luer needle so that I could bend it and experimented on the various shapes the needle must have, readily to pass up through the posterior palatine canal. The result was the needle, actual size cut of which accompanies this article, which Beckton, Dickinson & Co. have made for me.

Besides the tendency to strike the posterior wall of the canal, a straight needle tends to pass too far externally to enter the ganglion and it also naturally passes through the sphenomaxillary fissure into the orbit.

Sluder, who originated the injection of the sphenopalatine ganglion, employs a strong, straight needle which penetrates the posterior tip of the middle turbinate and passes on into the sphenomaxillary fossa. By this route, the needle necessarily cannot get far from the perpendicular plate of the palate bone. Therefore, to inject the ganglion per posterior palatine canal, one has to use a curved needle and its passage must be so that its tip travels upward and somewhat inward. In so doing, its tip naturally comes up against the bony roof of the fossa, just above the ganglion, instead of passing through the sphenomaxillary fissure. Of course, one must not insert the needle more than the 4 or 4.5 c.m., but it is a great help to have this bony roof as a natural boundary. The curved needle, therefore, passes most easily through the canal and comes most accurately in contact with the ganglion.

My needle can be used on any Luer syringe, but especially effectively on the B. D. Luer-lock syringe, illustrated. Not to be obliged to purchase a special syringe made with the shank bent for an angle necessary to use in the canal cuts down the expense of one's armamentarium.

My technique is to place a straight tongue-blade on the lower incisors, as a fulcrum, with the distal end pressed against the outer, posterior border of the hard palate. This keeps the tongue out of the way and orients one regarding the entrance to posterior palatine canal. Touch the roof of mouth in front of the blade with tincture of iodin. About ½ inch in front of the back edge of the hard palate, rather close to the alveolar process, one finds the entrance to the canal. Using a "finder" for this purpose is a convenience, but the point of the needle suffices. Then, holding the



syringe, needle attached, up against the upper, outer incisors of the opposite side, insert the needle by pushing it in, at the same time depressing the syringe until it comes against the lower central incisors, giving the syringe a slight twist in its descent in order to guide the point slightly inward and upward. In this way, somewhat as we were taught to pass a urethral sound, the needle comes up against the roof of the fossa and the injection is made. I have a scratch-mark at 4.5 c.m., as a precaution as to the safe depth to make the injection, but usually take the roof barrier as my guide.

Hoping that this may be of some help to those who practice sphenopalatine ganglion therapy, I am

Yours very truly,

W. D. CHASE.

230 East Broad Street, Bethlehem, Pa.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Regular Meeting, Jan. 8, 1926.

DR. J. MORRISSET SMITH, *Chairman.*

Demonstration of a New Instrument for the Treatment of O.M.C.C. with Overheated Air. Dr. John Guttman.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

DISCUSSION.

DR. BLACKWELL said in regards to Dr. Guttman's method of inflating the middle ear with hot air, he failed to see how the air would be warm when it reached the middle ear, for the reason it being a well known physical law that the temperature of air when under pressure becomes reduced, he himself had conceived the idea some fifteen years ago of doing the same thing recommended by Dr. Guttman, but became convinced after a series of experiments that the pressure necessary to inflate the ear refrigerated the air, due to this physical law.

DR. MAX UNGER said that he had studied the pressure necessary to inflate the ear and found that he could get into practically any ear with a pressure of one to two pounds. As to the compression cooling off the air, he understood that in this instrument the air was compressed before it was blown in and passed through the heating apparatus, so that no pressure was being applied to the ear. It is expanded, which would tend to make it still warmer.

DR. MARVIN JONES asked if the pressure *was* 16 pounds to the square inch, and expressed the opinion that 15 pounds of pressure would be more than sufficient to inflate the ear.

DR. BLACKWELL said that he had been doing it (15 pounds pressure) for the past fifteen years, and with the ordinary pump proposed by Mr. S.; usually the best results are obtained with 10 to 15 pounds, and he had never had a ruptured drum, or emphysema of the neck, or any other injurious feature following. In fact, the patients prefer the pump to the hand apparatus for the treatment.

DR. JONES thought that a pressure of 15 pounds in the cavity would be more than sufficient to rupture the drum.

DR. BLACKWELL replied that he did not know what was the pressure in the cavity, he only knew what was recorded on the instrument.

DR. JONES repeated that it would seem that 15 pounds of pressure in the cavity would be more than sufficient to rupture the drum.

DR. GUTTMAN showed the catheter employed and demonstrated the manner of using it. He said that he had not used any measuring standard for the heat, since the patient himself was a good enough measure and could tell whether it felt warm or not. When the heat was too great and the patient could not stand it, the air pressure had to be lessened. Furthermore, if one wished an objective instead of a subjective sign, inspection of the drum membrane would show the veins running down the upper margin of the drum membrane and along the anterior and posterior ligaments that were not visible prior to the treatment. That showed that the heat in the tympanic cavity distended the blood vessels. In many instances the patients were improved by this treatment and were relieved of the distressing noises. Heat is a good remedy in many affections, and why not in this?

Presentation of Patients Showing Postoperative Results Following Simple and Double Modified Radical Operations for Chronic Mastoiditis. Dr. H. B. Blackwell.

The six patients here presented, five of whom are children, have all been afflicted with chronic otorrhea previous to modified radical operation of from two to eight years' duration. Five of the operations were unilateral, and in one instance both mastoids were operated upon; all of the operations were

performed within the past year. All of the operations were performed at the New York Eye and Ear Infirmary and all show surprisingly good results in a class of patients notoriously difficult to maintain the necessary postoperative contact when once the postoperative wound is healed. The point which we wish to emphasize, which all of these patients show, is the excellent hearing now enjoyed by each patient in the ear operated upon. It is quite obvious that had a radical operation been performed instead of a modified radical, the hearing in each instance would have been very materially reduced, as it is each patient can hear a low whisper at several feet, and in each instance the family believe that the hearing has materially improved since the operation has been performed. This operation is performed primarily to conserve and if possible improve the patient's hearing in cases of chronic or subacute otorrhea and not primarily to produce a dry ear as the middle ear structures are allowed to remain *in situ*, so consequently in some cases there may be a slight mucus discharge, nonodorous and intermittent, more like an exudate from the middle ear mucous membrane, which has no surgical significance; the same operation was performed on each of these patients, namely, postaural incision, exenteration of the mastoid, taking down the posterior bony canal wall, as in a radical, removing the epitympanic ring in its upper two-fifths, but not disturbing the drum or middle ear structures, which are allowed to remain *in situ*. A new plastic meatal flap is cut in the concha, the posterior wound is sutured as in a radical, and subsequently the wound dressed per canal. In closing, we wish to indicate that the danger of causing facial palsy is much less than in a radical operation as the ossicles and drum are not removed. This greatly lessens the danger of facial nerve injury through intratympanic instrumentation.

DISCUSSION.

DR. H. M. SCHEER said that at the Manhattan Eye, Ear and Throat Hospital, Dr. Page was quite an enthusiast in following Dr. Blackwell's modified radical procedure and had aroused similar enthusiasm among some of the workers in the clinic there. Dr. Scheer said that he himself had operated on four cases in the past year following the same procedure and technique, and with the same good results shown by Dr. Blackwell. In one case there was a mucus discharge, eight months after operation, but one could look into the ear where the mastoid bone and epitympanic ring had been removed and see dryness there, though in the region where the perforation was and still remains one could see a mucous membrane discharge from a region of the mucous membrane in the middle ear. In the other cases the middle ear is absolutely dry, despite the fact that a prominent otologist said recently that he believed the modified radical operation to be a humbug. It does the trick, and should be performed by more of the surgeons.

DR. GUTTMAN said he had no desire to criticize the operation, but did criticize Dr. Blackwell's indication and reasons for doing the operation. He claimed that the advantage of the operation was that it improves the hearing. Why did he not give the exact record of the hearing before and after the operation? There are methods of doing this, and it would be interesting to have the exact data. Regarding it from the principle of acoustics, it was difficult to understand how the ossicles remaining in the tympanic cavity would improve the hearing. If you take an ordinary drum and make a hole in it, how much good will it do to have few ossicles in there; especially if they are also eroded? It was difficult to understand how from the acoustic standpoint the retention of the ossicles were enough to cause good hearing.

DR. KNIGHT said that in cases of chronic middle ear suppuration with practically no granulations or polypi in the tympanic cavity, he had performed a simple mastoid operation, which in most of his cases resulted in a dry ear and good hearing. In regard to conservation of hearing, these cases were parallel to those of Dr. Blackwell, for in both instances the ossicles were left *in situ*. It is well known that the radical mastoid operation does not tend to improve the hearing but, on the contrary, may make it worse. So any operative procedure which conserves the hearing should be welcomed. It would be interesting to know what success Dr. Blackwell has had with his cholesteato-

matous cases, and whether or not he ruled them out when selecting his cases for operation.

DR. M. H. KAIDEN: The indication for the radical mastoid operation is not to improve the hearing, which is frequently diminished by the operative interference, neither to stop the aural discharge which often continues after the operation. The only and real indication of this operation is in order to prevent the occurrence of an intracranial complication when such is imminent or threatening, as in marginal drum perforations with bone caries or cholesteatoma. The modified or conservative radical operation has its only indication, according to Bondy, when the perforation is in Shrapnell's membrane, indicating disease of the attic. In such a case the conservative radical operation, by leaving the drum proper and ossicles intact, will prevent complications and may increase the hearing. If the perforation is central, however, no radical operation, conservatiye or otherwise, is indicated, and I cannot see how a modified radical operation in a case of this kind will increase the hearing. It would be interesting to know if Dr. Blackwell has made any attempt to limit his modified radical operations according to these indications.

DR. MCCOY said that Dr. Blackwell was to be congratulated upon devising and pursuing this operation. When he first brought it forward some fifteen or sixteen years ago he was nearly drummed out of the camp for advocating an unsurgical procedure, and yet when one considers that the modified mastoid operation is a life-saving procedure, exposing and removing all the dangerous areas, exposing the tegmen tympani, the tegmen antri, the region in front of the lateral sinus, and leaves the middle ear alone in a case uncomplicated with labyrinthine symptoms, etc.

Dr. McCoy said that he did not understand Dr. Blackwell to claim that the hearing was improved, but only that much better results were obtained after this operation than after the radical operation. This operation removes all the danger points and leaves the ear in better condition, with the footplate of the stapes in place, and gives the patient a better chance to retain what little hearing he had before. Dr. Blackwell deserved congratulations for his persistence and on the beautiful results obtained from the cases shown.

DR. BLACKWELL said that the purpose of performing this operation is first to conserve and if possible improve the patient's hearing and, second, to permanently drain and aerate the mastoid antrum region, thereby eliminating the possibility of serious intracranial complications, which sometimes occur during the course of chronic otorrhea.

In regards to Dr. Guttman's request for the exact hearing prior to operation in these patients, Dr. Blackwell said he had purposely refrained from stating the degree of quantitative hearing each patient possessed prior to operation for the same reason that he has also refrained from expressing a similar opinion as to the amount of hearing each patient at present possesses, the reason being he does not feel that outside the human voice or whisper there exists a satisfactory practical method of comparison at least as far as the patient is concerned, he does not believe that sound produced by mechanical or electrical agencies, such as in cases of acumeter, watch or audiometer, can compare with the human voice or whisper as a satisfactory practical basis of comparison. The important thing about these patients is, after all, not so much what the hearing formerly was but what it is at present subsequent to the operation as the success of a modified radical operation depends upon the amount of hearing with which it leaves the patient as against a radical operation. My primary purpose in bringing these patients here is to have the members of the Section test the hearing in each one of them; those who have done so, I am quite confident, will agree with me that as far as the human voice and whisper is concerned these patients hear much better than they would have had a radical operation been performed.

The question has been raised in what class of cases of chronic otorrhea should the otologist advise the modified radical operation as against the radical operation for its relief, provided that there is any drum or ossicles left in a given case of O.M.P.C., we do not believe that the indications for the performance differ materially for the performance of either a modified or a radical operation. We do not feel that the presence of cholesteatoma is a counter-indication, nor do we think that the location of the perforation in the drum,

whether central, marginal or attic, is of much significance. The one clinical fact which we have observed in our patients is that having performed the modified radical operation and permanently drained and aerated the antrum region as we do in these cases, the middle ear, which may have been discharging for years, immediately begins to improve, large perforations of the drum, involving almost one-third of its total area, will heal and the middle ear makes a serious effort for the first time to restore itself to a more or less normal condition.

A Case of Jugular Bulb Suppuration Following Mastoiditis and Sinus Thrombosis; Operation; Recovery. Dr. John McCoy.

The following interesting case occurred in my service at the New York Eye and Ear Infirmary:

History: On March 30, 1925, Mrs. V. B., age 26 years, native of Finland, housewife, entered the hospital with the typical history of acute inflammation of the left middle ear and mastoid for the preceding three weeks, and with all the indications for opening and draining the left mastoid.

Operation: On March 30, 1925, a simple mastoid operation was performed on the left mastoid by one of my assistant surgeons. The pathology found at operation was that the mastoid cortex was very dense. Pus was found in the antrum and tip cells. A smear from the pus showed streptococcus. The sinus was far forward. Some dura was exposed in the middle fossa. The wound was lightly packed and partly closed. The temperature at the time of operation was 99.° Four days after the operation, on April 3, the patient felt chilly and the temperature rose to 104.° A blood culture taken at this time was negative. The blood count showed 4,800,000 red blood cells, 13,000 white blood cells, and 83 per cent polys. The temperature came down by lysis, and on the morning of the third day following was normal. That afternoon the temperature rose to 104.° In the absence of all other physical signs, the internal jugular vein was ligated at the level of the cricoid cartilage. The lateral sinus was then opened. It contained a clot and pure pus. Bleeding was established from the torcular end, but not from the bulb. Following this operation, the temperature fluctuated daily, reaching a low point of 99° and a high point of 105.° At each dressing, a small amount of pus welled up from the jugular bulb into the lower end of the mastoid wound. Blood cultures were taken on April 4, 6, 10 and 11. These were all negative. A blood culture taken on April 13 showed thirty-six colonies of streptococcus in twenty-four hours. We then decided to drain the bulb.

Operation, jugular bulb, April 14, 1925: Incision from the lower part of the mastoid wound to the point where the internal jugular vein had been ligated. The anterior edge of the sternocleidomastoid muscle was exposed and pushed back. The gland over the carotid sheath at the angle of the jaw was removed, and the internal jugular vein was ligated above the facial vein. The internal jugular vein was now exposed up to the bulb, and a search was made for the spinal accessory nerve. In this case, however, it was not found lying on the dorsal surface of the vein, but was afterwards found ventral to the vein. The styloid process was located and the posterior belly of the digastric muscle was incised and thrown forward to cover the exit of the facial nerve at the base of the styloid process. The finger was then passed into the jugular foramen, and the vein was separated from the margin of the foramen. The bone was then removed from the sigmoid sinus to the bony margin of the jugular foramen, and the sinus bulb and vein were split wide open. Following the bulb operation, the temperature did not rise above 102,° and the blood cultures taken the following day proved negative. On the fourth day following the operation, the patient complained of a pain in the right shoulder. No swelling or redness was observed, but the temperature rose to 105.° On the following day, April 19, the patient had a sharp pain in the left chest, with cough, and spit up bright red blood. There were no physical signs of a small consolidated area below the angle of the scapula behind. The temperature continued up and down for four days. The size of the consolidated area appeared to remain the same. On the sixth day there were definite signs of liquid in the plura, and an exploratory puncture obtained a few c.c. of thin seropus. Culture of this showed hemolytic strepto-

coccus. The wound in the neck was clean and healthy, so on April 26 she was transferred to the New York Hospital under the service of Dr. Poole. Her subsequent history showed that she had her chest aspirated three times, when an average of 500 c.c. of serosanguinolent fluid was removed on three successive days. On May 1 the right shoulder was aspirated and pus obtained, deep but extracapsular. On the same day, a thoracotomy was done under local anesthesia. The ninth rib was resected for a distance of 1½ inches in the posterior axillary line. Following these operations, the patient did well. Her temperature returned to normal, and she was discharged from the hospital, cured, on May 26.

Report of a Case of Chronic Middle Ear and Labyrinth Suppuration; Radical Mastoid and Labyrinth Operation; Recovery. Dr. John McCoy.

Mr. Leo R., age 36 years, single, native of United States. History: In October, 1909, he had an accident in a roller coaster which caused a fracture of the left mandible and lacerations of the head. He had, at that time, a decompression and was laid up for several months. In 1910 he developed scarlet fever and diphtheria with an acute abscess in the left ear and spontaneous rupture of the drum. The discharge continued until 1911, when a mastoidectomy was performed in a Philadelphia hospital. There has been an odorous discharge since that time. In 1923 dizzy attacks started with no nausea or vomiting. He had a tendency to fall forward and to the left. Since that time he has had five or six such attacks, lasting a few days each. On October 1, 1925, he had a severe attack of dizziness, staggering, nausea and vomiting lasting three days. When I first saw him about Oct. 10, at the New York Polyclinic, he showed the following conditions: The left drum was almost completely destroyed and there was foul smelling pus in the middle ear. The mastoid showed an old healed mastoid wound. Tests of his hearing showed that he heard absolutely nothing on the left ear. The Weber was referred to the right ear. The vestibular tests showed absolutely no response to caloric or turning tests. The fistula test was tried, but no result obtained. The patient was referred to the Neurological Department of the New York Polyclinic Hospital and they returned negative findings. A lumbar puncture showed a pressure of 12 m.m. The spinal fluid and blood Wassermann tests were negative. It was, therefore, decided that the patient had a chronic middle ear suppuration, with a dead labyrinth, and an operation was advised.

Operation: Oct. 16, 1925. Incision posterior to old mastoid scar. A number of old mastoid cells were removed. The posterior wall was taken away and a complete radical mastoid operation performed. A good-sized fistula was found in the external semicircular canal containing foul smelling pus. The vestibule and semicircular canals were opened freely and the first and second turns of the cochlea were removed. The wound was packed and later was dressed with Daken's solution until its complete recovery about seven weeks afterwards. He now feels perfectly well and all his symptoms have left him.

DISCUSSION.

DR. BLACKWELL said that Dr. McCoy's cases were very interesting and that he was to be congratulated upon the results. As regards his own attitude on this subject he now rarely feels disposed to operate upon the labyrinth when it becomes involved in cases of mastoiditis. He had recently had three cases of labyrinthitis developing in connection with mastoiditis, all three underwent mastoid or radical operations, two were cases of demonstrable fistula, nothing was done to the labyrinth at the time of mastoid operation or subsquently, all three patients recovered, and in two instances the hearing returned, having previously lost it completely. His views, therefore, in regards to operating upon the labyrinth have been much modified.

DR. KAHN, speaking of the first case, said that he was impressed with the fact that we had much to learn regarding the spread of infection from an infected ear. Many cases had distant involvement (especially chest lesions like the case reported) following mastoid disease, even after repeated blood cultures came back with a negative report. Dr McCoy's case was compli-

cated by a pleuritic effusion. Dr. Kahn recently had a case of pneumonia with pleuritic effusion, the lesion involving the right chest, following a primary right mastoid. The same germ causing the mastoid was found in the pleuritic effusion. Undoubtedly the infection spread downward through the jugular vessels. There was a temperature rise to 105° with a chill; but blood cultures on several occasions always remained negative.

Dr. Kahn said that he had no personal desire to bring up the old question of ligation versus resection of the jugular vein; but in any fair discussion of this case the question was bound to arise. He understood that in this case no bleeding took place from the lower end direction, when the sinus was opened, and yet the general system was only protected by a low ligation. He thought this procedure was a mistake. Either the jugular should have been ligated above the facial vein or a complete resection done.

Referring to Dr. McCoy's other case: The patient stated that he was not dizzy following the labyrinth operation. He had a dead labyrinth before the operation. He did not respond to any labyrinth tests. All labyrinth tests were negative. It was difficult to understand how the labyrinth operation affected his dizziness. How could destruction of a labyrinth already dead effect dizziness? It would be interesting to know how Dr. McCoy accounted for the fact that the dizziness disappeared after the labyrinth operation.

Dr. Kahn said further, considering labyrinth operations broadly; that he did not consider that the question of when to do operations of the Richard's type on the labyrinth was definitely fixed in the minds of present-day otologists. So many cases had recovered where only the radical mastoid operation was done and where the Richard's type of operation seemed to be indicated. On the other hand there were good recoveries when the operation had been done. He usually preferred doing only the radical mastoid operation. However, if a labyrinth operation could be said to be indicated he preferred opening it more radically than that done by the Richard's type of operation.

DR. MAYBAUM said that Dr. McCoy's cases were of undoubted interest. Such cases should be frequently reported before the Section as each one is not only a lesson unto itself, but of considerable importance in the diagnosis and management of similar cases.

The point that impressed him in listening to the presentation of the first case was the definite and clear indication for a jugular bulb operation. The patient had a persistent bacteremia after the sinus had been dealt with and drainage from the bulb was inadequate, *i. e.*, pus could be squeezed into the mastoid cavity through the bulb by pressure below the angle of the jaw. The point of importance for discussion is the type of operation advisable in such cases. Dr. Maybaum said that in his opinion the objection to the operation described by Dr. McCoy is that it is a procedure of considerable magnitude, entailing a certain amount of risk to patients who at best cannot stand prolonged operative interference. Only one thoroughly conversant with the deep anatomy of the neck should undertake such an operation.

Dr. Maybaum said that in the last few years Dr. Friesner and he had done a number of jugular bulb operations by a far simpler method. Some of the cases had been presented before this Section. This operation needs no proper name; it has some of the desirable features of the Grunert operation and is in reality an approach to the bulb by means of a complete sigmoid sinus operation. After its completion, in the majority of instances, one can look into the bulb and without any difficulty thoroughly curette it. Through and through irrigation from above with Dakin's solution can be done daily, a catheter having been placed in the bulb.

The operation is performed as follows: The fibers of the splenius capitus are separated from their attachment to the occiput; with an elevator the periosteum covering the occiput is detached. An assistant retracts the soft parts. The next and very important step is to expose the cerebellar dura below and behind the sigmoid sinus for a width of one-half to one inch. This is done so as not to injure the dura during the subsequent steps. The posteroexternal edge of the sinus groove is then removed. As one approaches the bulb the posteroexternal surface of the sinus groove becomes the posteroinferior surface. As a last step with a curved rongeur the

jugular process of the occiput is removed. This brings the bulb into view. Rarely the sigmoid sinus approaches the posterior canal at a rather sharp angle, making it difficult to uncover the bulb; the procedure should, however, be attempted before doing a more formidable operation.

From the standpoint of simplicity—but four or five minutes are necessary—this technique is worth trying. Also from another very important standpoint, at all times, by following the technique carefully, the facial nerve is free from danger of injury. As one approaches the bulb, one is working behind and internal to it. Furthermore, the cosmetic results are far better with this method. There is no need, as in the case presented by Dr. McCoy, of joining the mastoid and neck scar. Approach to the jugular bulb from below by means of deep neck dissection, entails a certain amount of danger of a spreading cellulitis.

The second case of Dr. McCoy, requiring a labyrinthectomy, is of importance, because of decision necessary as to the question of operative indications. The patient had a dead labyrinth. Spinal fluid cell count was normal. Not every dead labyrinth requires a labyrinthectomy; if there are evidences of necrosis present at the time of the radical mastoid operation the labyrinth should be opened by the simplest procedure, *i. e.*, Hinsberg operation. A dead labyrinth without symptoms for a considerable time showing no evidences of necrosis, does not require interference.

The cases which offer difficult problems for decision are the acute cases—the diffuse purulent manifest labyrinthitis. With a normal temperature and normal spinal fluid, the case can be carefully watched. On the other hand, if there is a definite progress in the symptoms as indicated by temperature, headache, increased cell count in spinal fluid, the question of operative interference comes to the fore. Labyrinthectomy during the very acute stage of the disease offers more danger of spreading the infection to the meninges than a policy of waiting for the condition to quiet down. In traumatic cases it is a better plan to do labyrinthectomy with the first evidence of an increase of symptoms. It is often difficult to decide just when to interfere and the wrong decision may be made with the best care and intention.

The interesting problem for decision as to question of doing a labyrinthectomy presented itself in Dr. Friesner's service at the Mt. Sinai Hospital about two years ago. A young lady gave a history of a chronic suppurative middle ear with repeated labyrinthine attacks. On admission she had a rigid neck, severe headache, temperature 105,° a nonreacting labyrinth. Spinal fluid cell count was unusually high, 6000 cells, mostly polys., with no bacteria. We decided not to interfere at this time for fear of converting a localizing process into a diffuse meningitis. The temperature and symptoms gradually subsided. At the end of ten days there was a slight rising temperature, and a beginning return of symptoms. It was then decided to wait no longer. A labyrinthectomy was done. The girl made an uneventful recovery and was presented before the Section some time later. It may well be said that an identical case managed in a similar way might develop a diffuse meningitis.

DR. HELLER, referring to the question of resecting the vein or not, told of a case of a man, age 32 years, who consulted him for an acute exacerbation of a chronic otitis media, although his ear had been dry for several years. His temperature was 102° or thereabout, and he had symptoms of an acute mastoid. He was operated upon a few days later, disclosing the regular mastoid findings. He did well for a few days, and then had a temperature of 106,° and ran an up and down temperature for three or four days, but all the blood culture reports came back negative. There were no other symptoms than this rise of temperature, and no chills after the first two. A member of the Section saw him in consultation, and it was agreed to open up the sinus and tie the jugular. Dr. Heller made an attempt to tie the jugular and could not find it. He found the carotid and pneumogastric nerve, but no jugular, tlthough he tried for thirty minutes. Down at the level of the thyroid gland was a bluish mass which seemed to be a part of the thyroid gland. A general surgeon who was present also tried to find the jugular, but could not. Accordingly that part of the neck was closed, and he proceeded to open the sinus, which was found to be perfectly empty. There was no blood obtainable from any part of it. He then reached the conclu-

sion, which was subsequently verified, that the patient had had a similar attack of acute otitis media with chills and fever and probably a sinus thrombosis, which nature had healed; and what was supposed to be a thyroid gland was a big varix, which fortunately had not been opened up. It was the dilatation of the upper end of what was left of the jugular. Of course, there was nothing to be done to the sinus. The operation did the man no good, so far as his general condition was concerned, and for three or four days he continued this temperature of 104° to 106.° Then an internist was called in to look at him, who made a tentative diagnosis of pulmonary infarct, and three or four days later he coughed up blood; temperature gradually fell, and he later recovered. That was the first case he knew of where the sinus had healed itself.

Dr. Scheer, referring to Dr. McCoy's second case, told of an instance in which a very interesting factor occurred ten months after operation. In 1924 an actor was on the road attending to his vocation and had a severe attack of rhinitis, followed in twenty-four hours by acute middle ear symptoms. He was very ill, with dizziness and vomiting for two days. When he reached the city here his symptoms had subsided. I saw him first two and a half weeks after the onset of the condition. He complained of moderate pain in the mastoid process, but his dizziness and vomiting had ceased. Testing showed that he had a dead labyrinth on the affected side, with a tender mastoid; and after consultation it was decided to open the mastoid, but not to disturb the labyrinth for fear of starting up trouble, hoping that the mastoid operation would relieve him, for the time being anyway, and that later on further operation could be done if required. The modified mastoid operation was done, with the idea that possibly the infection in the middle ear would be somewhat alleviated by the opening up and aereating of the upper regions of the tympanic cavity. The mastoid wound healed but the ear kept on discharging for ten months, although the patient did very well; but one day in cleansing the ear a probe was put into the upper region of the modified radical wound; I encountered a grating sensation; and on putting in a pair of forceps a sequestrum was removed which proved to be a part of the semicircular canal. One could see the lumen and could see that it was definitely a part of the semicircular canal. Within a year the ear was entirely dry, although it is now moist. While the performance of the modified radical operation probably warded off any intracranial complications, the question remained whether the condition might not start up again later on.

Dr. McCoy said that some of the questions regarding the dead labyrinth dovetailed. It is said to be dead when the patient does not respond to the turning or the caloric tests, but does that mean that the labyrinth is really dead? In his opinion, it does not; it merely means that the endolymph does not circulate and stimulate the nerve fibres. Personally, he believes that if the patient has a circumscribed labyrinth one is justified in doing a radical mastoid operation; but when there is a dead labyrinth, with foul smelling pus from the fistula in the semicircular canal, one cannot tell where the infection might extend, and then the old-fashioned free incision and drainage would seem to be the safe procedure, leaving the labyrinth wide open, rather than to take chances. We know that before the operation came into vogue many patients died as the result of labyrinthitis. Dr. McCoy repeated that his choice would be to open and drain. When he sees a dead labyrinth he believes that the nerve terminals are not dead and they would carry infection into the brain. He would rather take the chance of giving the patient the old-fashioned drainage.

As to the other case, it got through all right with simple ligation of the vein at the easiest point. Perhaps all have seen many patients killed by prolonged surgery, when the patients had shock from sepsis, etc. The less operating one has to do, the better the chances for the recovery of the patient. Our principle has been to ligate as quickly as possible, say about five minutes. If possible, have a blood transfusion, and get the patient back to bed as soon as possible, and then give more blood transfusions. In this way the patients are less sick and get well more quickly. In this particular case a mistake was made in ligating below the facial, and in future cases it

would probably be better to take a little more time and ligate above the facial. It might require five minutes more, but in view of one case like this it would seem best to do it.

As to the type of operation, Dr. McCoy called attention to the fact that in giving the history he had stated that the woman had a very far forward sinus. In a paper on this type of operation which he had read last spring before the Ear Section of the Academy, he had stressed the fact that this was a difficult type of case in which to approach the bulb. Dr. McCoy said he felt that the type of operation which Dr. Maybaum proposed was not so complete as the one he does. In the paper he had read last spring, he had stated that in his opinion it was well to first ligate the vein, and if symptoms appeared later on one could wash out the bulb from below. In this instance it was impossible on account of having a solid clot, so it was thought better to drain the bulb as described.

Successful Treatment of Chronic Discharging Ears and Allied Conditions by Means of Zinc Ionization. Dr. John McCoy.

DISCUSSION.

Dr. C. H. Smith said that he had begun this treatment a year ago and regretted that he had not been able to secure as good results as those reported by Dr. McCoy. In all, he had tried it on twenty cases, only one of which dried up, the other nineteen not improving. The method was tried on all kinds of O.M.P.C., though for the most part the cases were of the type spoken of by Dr. McCoy as best indicated, *i. e.*, with a large central perforation and weeping mucus surface, and in none of them was it successful, although precisely the same technique was observed. The case that did become dry was one where there was a very slight discharge, just enough to give a little crusting in two or three days after being dried out. The zinc ionization was also tried on two slow mastoid cases that were not healing; one of these healed after one treatment.

Dr. Goalwin asked for information on two purely technical points: First, why was the negative pole placed in the hand instead of placing it on the mastoid? The latter arrangement would direct the current so as to deposit the maximum amount of zinc in the deeper portions of the ear and mastoid.

Second, was the fluid ever removed and the amount of zinc salt left in it determined? It would hardly take 1 c.c. of fluid to fill up the external canal. Since a 1 per cent solution of zinc sulphate is used, 1 c.c. would contain only 0.01 gm. of the salt, and still less metallic zinc. How much of that is actually deposited in the tissues during each treatment?

Dr. McCoy, closing the discussion, expressed regret that Dr. Smith had not obtained equally good results as those reported, but that he himself would certainly recommend everyone to try the method. It would seem to be particularly useful in the cases spoken of by Dr. Blackwell where there was a long-continued discharge from the ear.

As for placing the electrode in the hand, very few patients would like to have a wet sponge placed on the neck. The local effect noted is a grayish deposit on the membrane, that can be plainly seen on looking in there.

Dr. McCoy referred to the fact that Dr. Smith had been successful with only one case out of twenty, but said that he himself had succeeded in fourteen out of eighteen cases, and from the results thus far obtained he would certainly advise a careful trial with it, for the patients were delighted with it. In some few cases where a radical mastoid had been performed, a few simple treatments completed the cure. That, however, is not all of it. If the ear is dried up, there is likely to be a recurrence if infections from the nose and throat start up after the treatment, so it must be followed up by a thorough cleaning out of the nose and throat.

Dr. Heller inquired why zinc was used instead of other metals.

Dr. McCoy replied that it was first used by Ledoux, in France, and then by Dr. Freel, who reported a large number of cases with excellent results. Dr. McCoy said he had not himself tried other metals, though others had done so, notably zinc iodid, which required a reversal of the poles, but apparently gave no better results than plain zinc sulphate in 1 or 2 per cent solutions.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, January 27, 1926.

Chronic Antritis, Necrosis of Superior Maxilla, Operation, Oral Fistula.
Dr. Irving Wilson Voorhees.

On Jan. 17, 1924, this patient, Mr. A. D., consulted his dentist, Dr. J. H. Kauffmann, of 110 East 54th street, New York City, for severe pain in the right upper jaw. Dr. Kauffmann has been good enough to supply me with the following notes for the purpose of laying the background of this case report.

There are many badly infected and decayed teeth. The entire mouth is in bad condition and shows only too well the evidences of prolonged neglect. There is a sinus leading from the molar area on the right side into the antrum through which bare bone is felt. Several teeth were extracted at this time, and on Jan. 24, under novocain anesthesia, a wide incision was made in the canine fossa, sufficient bone was removed to explore the antrum, which was curetted, and much pus and necrotic tissue were removed. No opening was made into the nose.

Following this operative work the antrum was washed out with warm boric acid daily, and on March 17 more bone was removed, followed one month later by the fitting of a rubber denture to keep food and bacteria from entering from the mouth.

On June 15 a wide palatal flap was dissected free under novocain anesthesia and sutured to the buccal mucosa; the sutures were removed one week later. Irrigation with boric was continued.

On July 5, a small sinus still being present, another attempt at plastic closure was made, but it was unsuccessful. The patient was seen on August 29, at which time he seemed greatly improved in general health, but the discharging sinus still persisted. So much for the dental history.

Nov. 5, 1925, he came to my office to get relief from the foul discharge, which at this time was also affecting his nose. The sinus extending up from about where the last molar had been was easily located and the antrum was determined to be partially denuded. There was no communication with the nose except through the normal antral opening. A small amount of polypoid tissue was present in the right middle meatus. X-ray examination showed both antra cloudy, but the right was fairly opaque.

The patient entered the Manhattan Eye, Ear and Throat Hospital, where I did a radical Caldwell-Luc upon the right antrum on Nov. 16, under general anesthesia. The gingival margin was found to be so necrotic that it was necessary to scrape away a large part of it before healthy bone was reached. While carrying this out there was rather sharp bleeding, and it was only after clearing away all blood and debris that a foreign body, which is apparently an imperfect unerupted molar, was found lying free in the cavity.

An attempt was made to close the opening leading from the mouth into the antrum, but this was only partially successful and the wound healed, leaving this rather large sinus through which I have been treating the antrum for some weeks until it is now nearly obliterated by granulation tissue.

As soon as the nose is entirely clean and free from pus and crusts a further attempt will be made to close the fistula by making a buccal flap and suturing it to the elevated and denuded palatal tissue. The result will be reported to this Society later.

DISCUSSION.

DR. ABRAHAM, supposing the alveolus to be completely gone, suggested a sliding process of the soft tissue covering the hard palate and attaching it so as to cover the opening. In a somewhat similar instance he had succeeded very nicely by so doing.

DR. CARTER thought that the opening should not be closed at present; the antrum has a very foul appearance and should be treated for quite a time with alkalin douches and other methods for causing healing before any attempt should be made to close it. The amount of suppuration the antrum will stand without necrosis of the bone is remarkable. Dr. Carter said he always regarded these cases with a great deal of suspicion and we should be

absolutely certain that there is no syphilitic infection present. Although the Wassermann report was negative, it is well known that many syphilitic cases will give a negative reaction, and it is possible that such infection might be present in this instance. It would be well for Dr. Voorhees to make a physiological test with iodid of potassium to try to determine positively that there is no syphilitic infection before any operative procedure is attempted. The best way to close the opening would be to throw a rather large flap over it from the buccal mucous membrane. That could be done if the flap was sufficiently thick to include the submucous blood vessels and large enough to avoid any tension. Of course after the mucous membrane flap is adjusted the edges of the area whence it was taken would have to be elevated so as to pull them as nearly together as possible. There seemed to Dr. Carter no reason why the opening could not be closed in this way, but no attempt should be made until it was more nearly healed and was found to be absolutely free of syphilitis infection.

DR. VOORHEES said that Dr. Carter had outlined the procedure he already had in mind; he had also thought of the palatal flap suggested by Dr. Abraham, but in his own experience it was a little more difficult to get a good result with such a flap than with the buccal flap. Another question is whether there would be undesirable adhesions and contractions at the angle from which the flap was taken, but that would have to be tried out. It would probably be a couple of months before further operation was attempted, but later on the case would be presented again to show the results —good or bad.

Clinical Report on the Use of Acriviolet in the Diseases of the Upper Respiratory Tract. Dr. Arthur Herzig.

DISCUSSION.

DR. J. W. CHURCHMAN said he would prove briefly some of the reasons from the experimental and clinical sides of the question for a feeling of encouragement as to the future value of dyes in the treatment of infections. The facts brought out were illustrated by lantern slides. The following characteristics of the dyes which they appear to possess in a degree not shared in by the commonly used antiseptics, are the properties of particular interest in the study of the treatment of infections, particularly surface infections. No dyes or any other antiseptic substances will kill bacteria which it does not reach, and it ought not to be necessary to say that the use of a dye in no way relieves one from the responsibility of applying to the treatment of an infected wound proper surgical principles.

1. First of all, the dyes are penetrative. This property may be readily demonstrated by experiments in animals, but I have also demonstrated it by observations on the human being. In the patient who was to undergo a mid-thigh amputation, the knee joint was injected after anesthesia had been induced, with gentian violet. After the leg had been removed the synovial membrane was fixed and sectioned by special technique and examination showed that the dye had penetrated to the subepithelial layers. The penetrative power was demonstrated still more strikingly by observations on patients undergoing colectomy. After the abdomen had been opened, a rubber tube was passed by an assistant per rectum high into the bowel, and through this tube gentian violet was infected into a loop of the bowel isolated by the operator between ligatures. The isolated loop was thus distended with dye during the course of the operation and immediately after completion of the colectomy the distended loop was properly fixed and frozen sections made of it. It was found that the dye had penetrated through the thickness of the mucosa.

2. The dyes are strongly bacteriostatic. They are capable of killing organisms by their power of inhibiting growth even in rather high dilutions. This is their most striking characteristic. This bacteriostatic power is selective and the selective feature is best shown by the use of gentian violet and other dyes of the chemical series. These dyes when added to the agar on which the organisms are planted, prevent the growth of many species but are without effect on many others. This selective power parallels very closely the Gram reaction. Other dyes, of which acriflavine in an example, exhibit a selective activity which is in some respects the opposite of that of

gentian violet, and the dye under discussion tonight, acriviolet, is a combination of two dyes which have somewhat different bacterial affinities. Acriviolet would appear to be slightly more effective than either of its components used alone.

3. The dyes have the power of preventing sporulation. In some types of organisms if a little acid fuchsine be added to a suspension of B. subtilis in a vegetative form, no sporulation will occur, though the bacteria are not killed. This is an interesting instance of selective bacteriostasis in which one function of an organism is paralyzed by a dye which appears to be without much effect on the other functions.

4. The dyes are relatively nonirritating. Statements of this kind must always be qualified and in the case of dyes it must first be said that in the urethra and nose, acriviolet at least must be used, if at all, with great caution. But in general, the dyes under discussion are in the strengths which strongly inhibit bacteria almost harmless to the tissues.

5. Clinical evidence is at hand that the dyes are actually capable of causing bacteria to disappear from infected surfaces. Two years ago, I had the opportunity to study this question under peculiarly favorable conditions in the treatment of a woman suffering from acute gonorrheal cervicitis. She had had the tubes ligated two years previously and we therefore had no fear that interuterine infections would make infection of the peritoneal cavity. The vagina, labia and urethral orifice were thoroughly painted with hot acriviolet and the uterine cavity was distended through a silver catheter. Daily microscopic examinations of vaginal smears showed that the dyes caused prompt disappearance of all bacteria. These, of course, would not stay away, reappearing when the dyes were omitted by reinfection from without. The gonococci would, however, stay away permanently. It is not to be presumed that the result obtained in this particularly favorable case could be duplicated in the ordinary case of cervicitis. But the examination did provide an opportunity to demonstrate in a living human being the bacteriostatic power of the dye.

These are some of the reasons which justify the hope that extensive study of the questions discussed now under way in laboratories and clinics will lead to an advance in the treatment of infections.

DR. JOHN MACKENTY: My experience in the use of acriviolet, in acute rhinopharyngitis, has not been favorable, as I have observed that it many times causes irritation in this condition.

In acute follicular tonsillitis it is of distinct value, when applied frequently. In its use I instruct the nurse to apply the dye every hour or two, using 1 in 200 solution.

In chronically diseased tonsils, it may have some value in preventing acute attacks if applied daily.

In Vincent's infection I cannot give any positive statement, since the disease varies so much in its duration. Many cases of Vincent's clear up in a few days on any treatment, or no treatment.

I use the dye for disinfection of the mouth, nose and hypopharynx, before laryngectomy, considering it better and less irritating than iodin.

I also use it after tonsillectomy in all cases, until the tonsil fossae are healed.

I have not employed it in any disease of the nose or accessory sinuses, so cannot give an opinion on this point. Theoretically, it should be of value in clearing up diseased conditions in the antra, if they have been widely opened, especially chronic conditions.

DR. ABRAHAM thanked Dr. Herzig for bringing before the Section the results of his work with this valuable remedy, and also Dr. Churchman for introducing the use of the dye in this country, his first experiments beginning in 1912 with gentian violet.

He then read an extract from an article by Faber and Dickey, of San Francisco, published in the *J. A. M. A.*, Sept. 19, 1925, on "The Treatment of Thrush with Gentian Violet," showing its value in checking an epidemic and use as a prophylactic; and also a report of its value in the treatment of ear infections, by G. J. Pearson, of Burlington, Iowa, after the method reported by Dr. Abraham in a paper read at St. Louis.

The canula spoken of by Dr. Herzig is the original Hartmann canula, so modified that without using the rubber attachment one can syringe directly by locking the canula to the syringe. Dr. Herzig also said that I had used the dye for four years; that is not quite correct, for in the beginning I used gentian violet.

Take a given furuncle that presents itself in a stage where it is swollen, red and tender, but has not gone into suppuration: cover the surface beyond the red with vaselin, and the dye will not spread beyond that. Make one or two applications of the dye with a cotton applicator, and in the majority of cases the furuncle will disappear without going on to suppuration. If, however, the funcle has already suppurated, it must be opened and treated accordingly.

Then a few points about the use of the dye. I have not changed my armamentarium. Many of the men object to the use of the acriviolet on account of the staining. I have a running cuspidor, as most men have, and keep on hand a 3 per cent hydrochloric acid solution, which takes off the stain promptly. Having this running water, I use the dye freely. Dr. Churchman spoke of the bacteriostatic action of the dye. To simply spray the patient's throat and let him go, will do no good. The dye must be in contact with the germ; it destroys it in about two minutes. The best results have been obtained with streptococcus infections of the throat, larynx, trachea and even of the bronchi. In the morning I make up a solution of two tablets in 30 c.c. of water; it is better to use the solution warm than cold. I then cocainize the pharynx and larynx with a weak solution, spraying it deeply into the larynx to stop the reflex cough; teach your patient to breathe in slowly and deeply. Then take the 1/100 solution and add an equal amount of hot water; cover the patient's lips and chin with vaselin. Now your patient should breathe in while you are spraying the dye, so that it will be deeply inhaled into the bronchial tubes. In these streptococcus infections it is surprising to see how those patients who breathe deeply will expectorate the dy in a few minutes, and you will get not gentian violet dye, but it will come up yellow, showing that the gentian violet has been used up, leaving the acriflavine. One gets remarkably good effects using it that way.

Now in tonsillitis, it is not the application on the tonsils, but in the crypts —by using the suction apparatus and then applying the dye in the crypts.

First, suction under cocain, removing the liquid pus; wash off the surface with an alkaline solution; teach the patient how to gargle; spray in a sufficient amount, etc. Repeat the performance, for two minutes, keeping the dye in contact with the crypts. In cases with glandular enlargement and with high temperature, it is surprising to see the reaction when the dye is used properly.

Now there is an objection to using the dye in rhinitis in strong solution as it may cause rhinorrhea; therefore you should use weaker solutions, etc. Otherwise make a local application.

In sinus disease: We have with us tonight a young man who has done all my bacteriological work, and he could tell you the effect of the dye. In a given case, for instance, an antrum that presents complications and does not heal under local treatment, in a case of that kind washing out the sinus with a simple alkaline solution, 2 per cent, follow that with plain water, and then inject a sufficient amount of dye into the antrum and withdraw the needle and leave as much of the dye as possible; take a culture before using the dye, and get your report on it; then take a second culture. In a few cases after injection of the dye and one syringing, the secretion from the cavity will show a predominating gentian violet of acriflavine color. This will aid you in making a diagnosis of either a gram positive or negative infection. After the second injection, and often after the first, you will be surprised at the result. Your dye is bacteriostatic. The patient's symptoms will disappear shortly after using the dye, but that does not mean that the sinuses will heal up. After washing the cavities three, four or five times, it has been my experience that the cases require an astringent treatment afterward.

DR. HAYS: After Dr. Abraham's declamatory and enthusiastic remarks, it hardly seems advisable to say anything of a derogatory nature in regard to the use of this dye. I am glad that he stated that he has been using the

dye less than four years; for some time before that, I read Dr. Churchman's paper and had a combination of gentian violet and acriflavine made up in ¼ gr. capsules by my druggist. So it appears that we had used the dye before Dr. Abraham did.

It is questionable to my mind whether we are getting any better results by the use of these new antiseptics in acute conditions than we did before we knew anything about them. Only the other day an out-of-town doctor came to the office and saw us using this dye, mercurochrome, the quartz lamp, etc., and asked, "When you come to it, are you getting any better results in your acute cases by the use of these things than you did before you knew about them?" I had to answer that I did not think so. Which reminds me that Dr. Abraham was very enthusiastic about the use of silver nitrate in sinus conditions before he knew of acriviolet.

From the scientific and laboratory point of view, there is no doubt of the value of the facts which Dr. Churchman has brought to us tonight. Two factors must be considered. In the first place, one cannot judge of the value of a medicament of this kind in acute cases, because they will recover in a short time anyhow. In the second place, one must consider the type of organism which is causing the infection and the resistance of the individual. Much as I dislike to limit the use of a product of this kind, I cannot help feeling that acute cases will do very well with ordinary cleansing and stimulation. When it comes to the chronic cases, there is no question of its value, provided the mucosa is thoroughly cleansed beforehand.

There are two facts which should be emphasized. First, the dye must be made fresh daily; and secondly, one should be sure that it is directly applied to the diseased part. Indirect application will be useless. I was particularly interested in one remark of Dr. Herzig, that he was able to get some of the acriviolet through an incision in the eardrum so that some of it will run down the Eustachian tube into the throat. It has been my experience that one can seldom get anything through an incision in the drum so that it will reach the middle ear. Moreover, in these acute cases, the Eustachian tube is so swollen that it does not seem reasonable to feel that any solution which is actually in the middle ear can get through it. However, if the facts are as Dr. Herzig states, I am sure we have an exceedingly valuable adjuvant to treatment. Dr. Herzig has certainly presented the subject in a very interesting and convincing manner.

Dr. Lederman said that he has used this dye and one great objection, which Dr. Abraham had spoken of, was the esthetic effect on patients who object to have the mouth, teeth and ears stained. Dr. Churchman's presentation also was most interesting, but if we are going to get therapeutic action from any remedy, the first and most important point is the thorough cleansing of the parts so that the medication will come in contact with the desired area. He further said that he had himself worked along these lines for some years with a few remedies, one of which, cresatine (Sulzberger), a metacresol preparation without escharotic action. This excellent preparation had proved very effective in furunculosis of the ear and other infections of the nose, throat and ear. It not only gives an antiseptic effect but has also an analgesic action and is almost a specific in cases of staphylococcus infections like furunculosis of the ear. It is not a new remedy, as it has been used for a number of years, and has proved very efficacious and does not stain the tissues. For ten years he has used another preparation which is a very active antiseptic without the disadvantage of discoloration: the iodin powder (Sulzberger). This has been used with much success in the treatment of nose and chronic suppurative ear conditions. In the latter conditions, douching is contraindicated, and this dry treatment has given excellent results when properly carried out. Dr. Lederman said he had seen many cases treated by skillful men, who had not taken the time necessary to clean the parts thoroughly before applying the remedy; but by taking this precaution this remedy, which is an iodoboric acid, in which the residue of boric acid remains for a short time after the free iodin has been given off, also is an iodized product and very soluble in the secretions, had given most satisfactory results. There is no doubt that acriviolet has bactericidal properties, but it does produce considerable discoloration of surrounding tissues, and thus might mask the clinical picture in acute conditions.

Intubation of the Esophagus for Carcinomatous Stricture. Dr. M. C. Myerson.

Because cases of carcinoma of the esophagus are almost all seen too late for radical surgical treatment, which is our only hope for cure, we are necessarily confined to methods of palliation for this condition.

Palliative treatment consists of measures to maintain the patency of the natural pathway to the stomach or of measures which disregard this pathway and introduce food directly into the stomach. Under the former type of treatment are to be mentioned dilatation as is practiced principally by Vinson and Moersch, or dilatation and intubation as is practiced by Symonds, Guisez and Souttar. Gastrostomy is practiced universally, perhaps so because the value of dilatation or dilatation and intubation is not well known. Vinson and Moersch have demonstrated the comparative safety and efficacy of dilatation of these carcinomatous strictures. Symonds has been intubating these unfortunate patients for the past forty years.

The method advocated is intubation of the strictured esophagus after dilatation has been carried out with the Plummer bougies passed over a previously swallowed thread. For intubation oval tubes of solid sterling silver are used, whose length vary from 3 to 4½ inches, and whose cross-section is 10 m.m. x 6 m.m., and 15 m.m. x 10 m.m.

Dilatation and intubation was first used by Krishaber, of Paris, in 1881. Hill was the first to dilate and intubate these strictures with the aid of the esophagoscope. In 1920 Lynah reported the use of silver and rubber tubes in upper esophageal carcinomata. Souttar has been using flexible, spiral-wound German silver wire tubes for some time.

The tube is implanted into the dilated carcinomatous area, and its position is confirmed by Roentgen study. The tube usually stays in place well. If it becomes displaced, it can be readily adjusted through the esophagoscope.

Four cases are reported, the longest duration of placement being sixty-nine days. Another patient has been wearing a tube for forty days and is comfortable. These patients can eat semisolid food and enjoy the privilege of the family table. Although they succumb to the carcinoma, they have the advantage of a patent esophagus until the end.

Dr. Herzig, replying to Dr. Hays' inquiry as to how he could force the dye through the opening in the tympanic membrane into and through the swollen Eustachian tube, stated that it was impossible to do so, but after twenty-four to forty-eight hours he could force some through. He also stated his belief that he has prevented several cases of acute otitis from developing into mastoiditis. He prepares the dye fresh for each case.

He agreed with Dr. Lederman as regards thoroughly cleansing of the parts prior to application of the dye, as Dr. Churchman had proven that the dye penetrates only when the mucus coating has been removed.

Dr. Hays said that Dr. Myerson had made a most excellent presentation of the subject, and if he himself had a carcinoma of the larynx and wanted to live any length of time he would certainly prefer the method presented rather than any other procedure. As a rule, the operations on the upper part of the esophagus have been more satisfactory than those on the lower portion. It would be interesting to know Dr. Myerson's views on that point.

Dr. Myerson in closing the discussion said that Dr. Torek has a patient upon whom he operated in 1913; he resected the esophagus and united the upper esophageal end with a gastrostomy opening by means of a long rubber tube. That was thirteen years ago, and the patient is still living. She is a lady about 70 years of age. Theer are not more than five or six brilliant surgical successes reported. Lilienthal, Eggers and Hedblom to my knowledge are credited with such successes.

The trouble is that practically all of these cases of carcinoma of the esophagus come under observation when metastasis has already taken place, or when the disease has extended so far that surgery is out of the question. The middle thoracic portion is considered the most suitable for surgical resection, and practically all the brilliant surgical results have come from operations on the middle thoracic esophagus. The exception to this, is the case of Hedblom, in which he resected the lower esophagus and stomach in several stages with a final cure. But, as a rule surgery is hopeless, for the cases are not seen early enough.

BOOK REVIEW.

Handbuch der Neurologie des Ohres. Professors G. Alexander and O. Marburg. Volume III. Urban & Schwarzenberg, Berlin, 1926.

Part II of this comprehensive classic of otoneurology presents a number of important special monographs. Special attention is given to, VI, tumors of the cochlea, vestibular system, brain stem and cerebellum. This chapter, prepared by so eminent an authority as Professor Marburg, of Vienna, constitutes one of the features of this volume.

There follows the operative therapy in brain tumors, by Professor Denk, of Vienna, and Roentgenology of diseases of the ear, including brain tumors of the posterior cranial fossa, by Dr. Sgalitzer.

VII. Dyskinesia and Dystonia, including a discussion on tonus; vestibular reactions and their relation to motor impulses, the significance of the pathology of the peripheral vestibular apparatus for tonus, motor disturbances in affections of the cerebellum and extrapyramid diseases. This chapter has been prepared by Dr. E. Pollak, of Vienna.

VIII. Neuroses and Psychoses. The cochlea of the labyrinth in relation to neuroesthenia, hysteria, migrane, etc., by Drs. Liedler and Loewy; Epilepsy, by Professor E. Raimann. The posthumous chapter on otogenic reflex neuroses, by the late Professor Urbantschitsch, of Vienna, is concluded in this chapter; Traumatic Neuroses by Professor Raimann; The Ear and Psychoses, Nystagmus, by Professor Ohm; Seasickness, by Dr. H. Abels; Experimental Analysis of Vegetative Reflex Action on the Labyrinth, by Dr. E. A. Spiegel; Spasmus Nutans, by Professor Zappert; Nerve Function and Nerve Disturbances in Aviation with special reference to the eighth nerve, by Dr. Palthe, constitute Chapter VIII.

IX. Dysglandular Diseases concludes this volume and includes a very valuable monograph by Professor G. Alexander on "Neurology of the Ear in Kratenism and Myxoedema, which exhaustively treats of the anatomy, functional tests, speech, diagnosis, treatment and final outcome of these conditions.

The final chapter on gland diseases and the organ of hearing, is prepared by Dr. J. Bauer.

To attempt an exhaustive review of this series of monographs would hardly fall within the scope of this notice. Suffice it to say, that the undertaking of the authors, Professors Alexander and Marburg, of Vienna, to publish so complete a treatise on otology and its relation to neurology, has met with the most unqualified endorsement on the part of the medical profession specializing in these fields. Volume I, which was issued less than a year ago and previously reviewed in these columns, has been enthusiastically acclaimed as one of the most important contributions to modern otological literature.

Conducted under the editorship of Dr. Hans Brunner, of Vienna, many phases and bearings on otology to neurology have appeared in well-ordered sequence and reflect the earnest efforts of investigators who are concentrating their energies on the highly specialized work which they have contributed to this special series of monographs.

No otologist of today, who recognizes the important advances which have been made in the investigations of the labyrinth, cerebral localization and neuropathology, can afford to be without this monumental work on neuro-otology. Two volumes have now appeared from the press of Urban & Schwarzenberg, and the third volume, which is now in preparation, will conclude the set.

The typography is perfect, the illustrations are remarkable for their clarity and quality, and the literature from which all of this data has been prepared is appended in ready-reference form at the conclusion of each monograph.

M. A. G.